CLINICAL SURGERY

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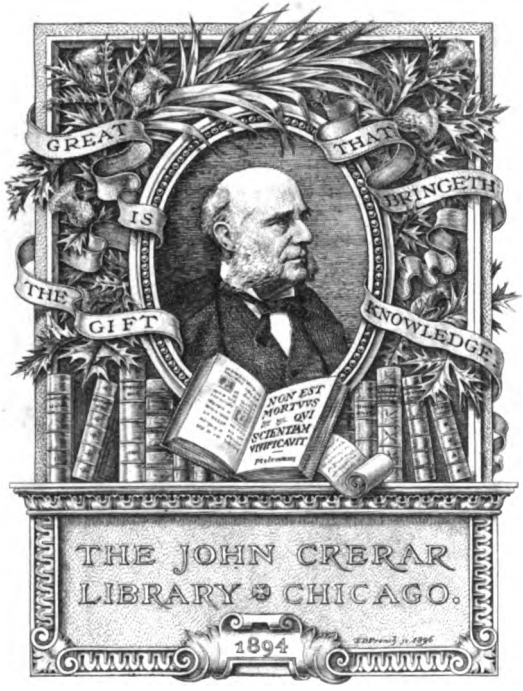

ATLAS

OF

CLINICAL SURGERY

WITH SPECIAL REFERENCE TO

DIAGNOSIS AND TREATMENT

FOR

PRACTITIONERS AND STUDENTS

BY

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ENGLISH ADAPTATION

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With 150 Colored Figures

From Models by F. Kolbow in the Pathoplastic Institute of Berlin.

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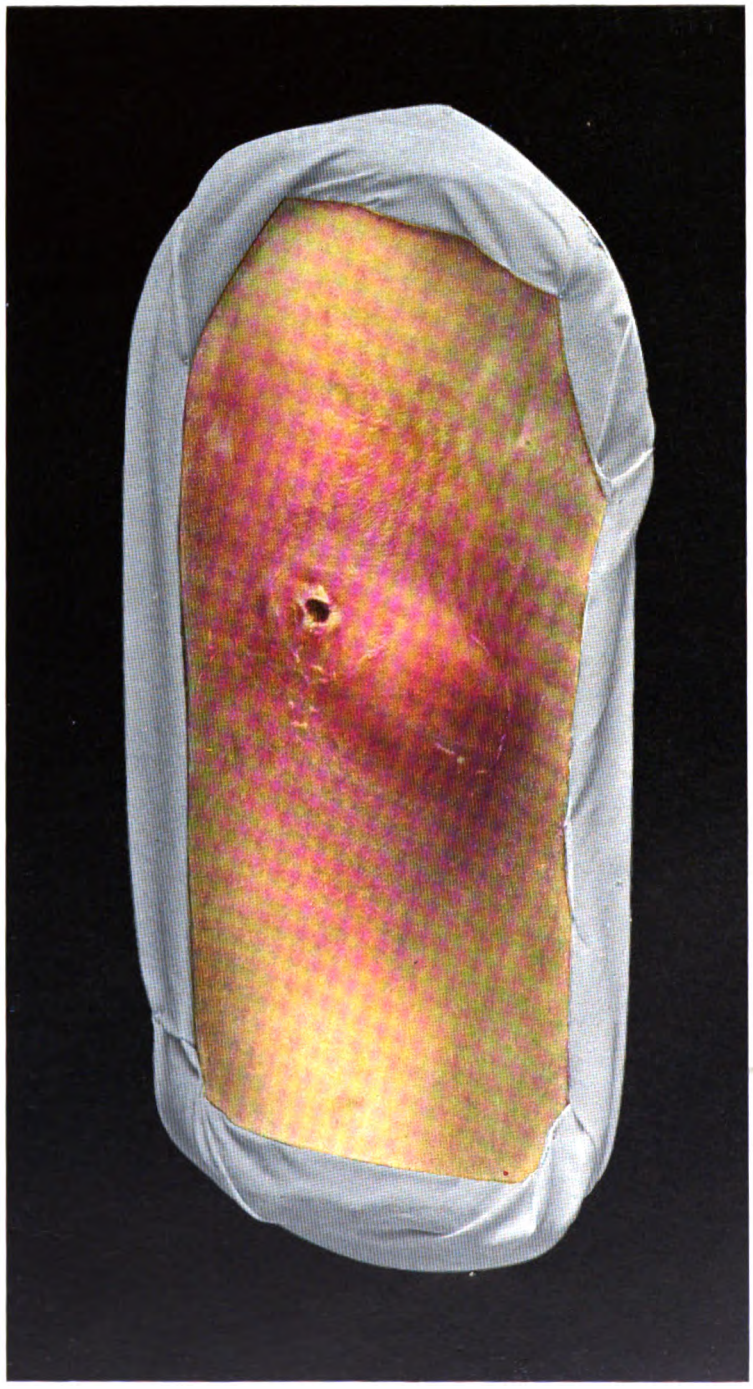

Fig. 108. Infectio generalisata.

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INFECTIO GENERALISATA (*General Infection*)

Plate LXXXVI, Fig. 108.

In the description of the various local pyogenic infections, mention has already been made of general infection. In every pyogenic and putrefactive infection there is a certain degree of general infection, but this is not generally sufficient to be recognized clinically or bacteriologically. In apparently benign pyogenic affections, such as furuncle, bacteria may be found in the blood. This explains the occasional occurrence of metastatic osteomyelitis in connection with such affections; and also the fact that the impairment in general health is often out of proportion to the local inflammation.

General infection assumes different clinical forms, but it is impossible to make a classification of these which is free from objection. Moreover, such a division is of little practical value, as the same measures must be employed against different forms of general infection. From the clinical point of view, it is, therefore, best to speak only of general infection, and abandon the old, and often inappropriate, terms sepsis, septicæmia and pyæmia. In any case the term sepsis should be confined only to that form of general infection which is caused by the putrefactive bacteria; but this form is rare, and it is generally a question of mixed infection with putrefactive bacteria and streptococci.

Again, the distinction into metastatic and non-metastatic general infection, proposed by *Lexer*, is practically without value and does not hold good for all cases. In many cases non-metastatic cannot be distinguished from metastatic general infection,

especially as both often co-exist, or one may merge into the other. Lastly, when only small metastases are present in the internal organs, and they remain unrecognized, such a metastatic form may be wrongly regarded as non-metastatic.

Bacteriological research has shown that general infection is not due to one specific cause; staphylococci, streptococci, pneumococci, typhoid bacilli, bacterium coli commune give rise to *pyogenic general infection*, while putrefactive bacteria (proteus vulgaris, etc.) cause *putrefactive general infection*. These two forms are often combined, and clinically indistinguishable, so that the designation general infection is sufficient for practical purposes. On the other hand, the nature of individual cases should be made clear by bacteriological investigation.

It has been shown that in general infection caused by staphylococci there are usually metastasic formations (ninety-five per cent. according to *Lehnartz*). Local infection with staphylococci is generally circumscribed, while streptococcal infection is more diffuse. This may depend on the fact that the staphylococci are accumulated in large masses, but it has not been proved.

In streptococcal general infection, on the other hand, there are hardly ever any metastatic formations. As cases without metastases are clinically the most severe, and almost always fatal, a division into metastatic and non-metastatic general infection is identical with less-severe and more-severe infection. However, just as infection with very virulent staphylococci may be fatal without metastatic formation, so may infection with less virulent streptococci cause metastatic formation and end in recovery. In practice, we know that streptococcal local infection is more severe than staphylococcal, and this usually holds with general infection.

It is impossible to introduce the ideas of bacteriæmia and toxinæmia into clinical nomenclature, for

the characterization of general infection. Although general infection may exist without the presence of bacteria in the blood being capable of demonstration by the present methods of bacteriological research, bacteria are no doubt present in the blood in every case of general infection, but are quickly destroyed by the bactericidal substances. On the other hand, bacteria may be found in the blood in cases of general infection in which toxinæmia is not recognizable. If toxins are always present in the blood, they are not easy to find, especially as they have a tendency to form combinations with the organs. In general infection there is always bacteriæmia and toxinæmia, but in practice we only speak of general infection which is most often acute, rarely chronic.

Acute general infection may be primary or secondary; mild or severe. The severity of general infection depends on the number and virulence of the bacteria, and on the power of resistance of the body. The severest forms of general infection appear so rapidly after the local infection that the latter remains in the background; these forms are often fatal before the typical inflammatory processes have developed at the seat of infection. Such cases include those which often occur in doctors from infection during operations or post-mortem examinations (streptococcal infection); also from infection by putrefactive bacteria; or by mixed infection by a symbiosis of streptococci and putrefactive bacteria. To this class belong cases formerly called cryptogenetic pyæmia, which are better considered as latent general infection arising from unrecognizable foci of infection. Virulent bacteria must often invade the intact mucous membrane and give rise to general infection.

In the great majority of cases, however, general infection is of gradual onset, arising from a local infection; but it is often in an advanced condition before it is recognized. It may occur by direct

extension of suppuration, or may arise without further extension of the local disease.

The more rapidly virulent bacteria enter the blood from the local seat of infection, the more severely is the organism affected. This is shown in the temperature chart.

In the most severe forms the temperature rises rapidly and remains at 40° or 42° C. (104°–107° F.); such cases generally cause death in a few days without metastatic formations. In less severe forms the temperature does not remain high, but is intermittent. This may be due to the intermittent entry of the bacteria and their toxins into the blood from the seat of infection, or to smaller quantities of them. When the organism conquers the bacteria and their toxins by the formation of antitoxin, the temperature falls; when the bacteria gain the upper hand the temperature rises.

The longer the process continues, the more frequent are the rigors, with intermissions of temperature. When these variations in temperature follow each other rapidly (as occurs in the severest cases) the temperature becomes continuous, the rigors cease and there are no metastases. If the organism gains the upper hand, the infection expends its energy in the formation of local metastatic formations in various places. This, in a way, may be regarded as a victory of the organism over the bacteria.

In the milder forms of general infection we therefore find metastatic formations in those parts of the body which are specially constituted to absorb bacteria and render them harmless (peritoneum, pleura, endocardium joint cavities). Metastatic formation is to be regarded as a curative process, as the bacteria are to a great extent destroyed. These metastases caused by bacteria in the blood must be distinguished from metastases propagated from purulent thrombophlebitis, or emboli containing bacteria. In all these cases the blood-stream plays the principal part in

general infection, the rôle of the lymphatics being subordinate.

As in local infection, general infection is predisposed to by debilitation of the organism by exhaustion, hunger, and exposure to cold, and by diseases such as diabetes and tuberculosis, etc. Along with general predisposition, there is a local predisposition depending on the nature and seat of the lesion. Foreign bodies often lead to general infection, also machine-injuries, compound fractures, bites, and wounds of the mouth and rectum.

The deeper the infection and the greater the pressure on the bacteria, the greater is their virulence, and therefore the more frequent is general infection. This accounts for the frequency of general infection in deep suppurations, such as those under the cervical fascia, and in the bones and joints. It is well known that the internal surface of the uterus during the puerperium is especially liable to infection, which may become general, and that the retention of pus and blood effusions are dangerous. Lastly, the treatment of infected wounds with strong caustics, such as carbolic acid, may give rise to general infection.

As regards the clinical symptoms of general infection, various clinical pictures may be produced according to the kind of infection, but the morbid condition is uniform as regards its most essential points. Bacteriological examination must decide which bacteria have caused the infection, whether one or more different kinds are present, and whether they are present in the blood (bacteriæmia). As regards metastases, we can only speak of metastatic general infections when metastases are found during life; while cases in which no metastases are found cannot be called non-metastatic till the absence of metastases has been established by post-mortem examination. Small metastatic foci are often found post-mortem (especially in the kidneys), which were not recognizable during life.

The symptoms of general infection differ according as the onset is sudden and acute, or gradual and chronic. In the most acute forms the symptoms appear suddenly, while in the other forms there is a latent stage with disturbances in the general condition (insomnia, loss of appetite, headache, pain at the seat of infection) which are premonitory of general infection. A frequent small pulse points to the onset of general infection, before the rise of temperature. The temperature then rises suddenly to 39° or 41° C. (102°–106° F.), with rigors. We have already pointed out that in the most severe cases, in which numerous virulent bacteria remain in the blood, the body is only able to offer a slight degree of resistance. In these cases there is no fall in temperature and no formation of abscesses, and the infection is often fatal in twenty-four hours or a few days, with high, continued fever.

On the other hand, if the bacteria only enter the blood intermittently, there may be periods during which fever is absent (*e.g.* after evacuation of retained virulent secretion by incision). With fresh infection of the blood there is at the same time a rise of temperature. Hence the variations in the temperature chart. Although remissions in temperature are characteristic of mild, general infection, this remittent fever after some days may become continuous and fatal. For example, when an extremity has been amputated for progressive suppuration, the temperature falls; but it may rise again after a time, showing that the organism was already saturated with bacteria and their toxins, and that the operation was performed too late to save life. It is noteworthy that the pulse in remittent fever remains small and rapid during the fall of temperature, even after complete cessation of the fever, showing how much the heart is affected by the process.

If, after extensive operative interference, the temperature approaches normal, this may be regarded

as a good sign for the further progress of the case. A subnormal temperature is sometimes observed in the most severe cases of general infection, and signifies complete collapse of the organism.

The respiration is rapid, as in all feverish conditions, and may become stertorous in severe cases with loss of consciousness.

Besides sudden rise of temperature and rapidity of the pulse, the tongue shows conditions which are characteristic of general infection. Changes in the tongue are observed even in slight disturbance in the wound. The tongue is at first smooth, dry and salmon colored; later on it becomes rough, fissured and brownish black. In severe cases of general infection the teeth are also dry and coated with sordes. The conjunctivæ are yellow, and in severe cases there may be jaundice of the whole body (hematogenous icterus). The patients are continually tormented by sweating and thirst.

These characteristic symptoms are diagnostic of a general infection whose point of origin is concealed. On the other hand, in general infection arising from infected wounds, the earliest signs pointing to general infection are often observed in the wound itself. As every pyogenic condition may lead to general infection, the wound must be continually watched by frequent changing of the dressings. The experienced can often foresee the onset of general infection, from the appearance of the wound. Apart from cases of general infection following a slight abrasion of the skin or mucous membrane, the wound generally becomes painful and œdematous; the granulations become unhealthy and flabby; the discharge of pus subsides and gives place to a scanty, dirty, often fetid secretion; the surface of the wound becomes dry and often covered by diphtheroid membrane (Fig. 101). Retention of pus, necrosis, extension of suppuration, lymphangitis and lymphadenitis are often concomitant signs. In infection by putrefactive

bacteria (Fig. 109) there are bullæ in the infiltrated skin and crepitation due to the formation of gas, and bubbles of gas in the secretion. Unfortunately, these characteristic signs are often overlooked; operative interference which could prevent extension of the already commencing general infection is neglected, and the condition passes into a stage which is almost always incurable.

In no other condition is the organism so much altered as in advanced general infection, so that the clinical symptoms become indelibly imprinted on the memory of the observer. All the already-mentioned symptoms of commencing general infection become intensified in advanced cases. The patients at first become light-headed, then delirious, and finally unconscious. The indifference of patients in the advanced stage is in marked contrast to their feeling of fear in the early stage of infection, and is an unfavorable sign. In the final stage, shortly before death, if the patient has not permanently lost consciousness, he often has attacks of fear, or even maniacal attacks, followed by collapse. In this stage the patient can hardly be kept in bed, as he makes repeated attempts to go home, etc.

The gastro-intestinal canal is severely affected; vomiting of blood from submucous hemorrhage, vomiting of bile and uncontrollable diarrhea result from the action of toxins. The skin is pale and cold, and may present morbilliform eruptions, erythema, erysipelatous reddening, vesicular eruptions, punctiform hemorrhages or more extensive blood-effusions. Bedsores are also common.

Almost all the internal organs are saturated with bacteria and their toxins, and react in their special manner. Nephritis is manifested by albuminuria; meningitis gives rise to stiffness of the neck; pleuritis causes blood-spitting; pericarditis is manifested by pericardial friction, and endocarditis (which is very common and often ulcerative) by cardiac murmurs.

As in every severe infection, the spleen is enlarged, and sometimes there is acute bronchocele.

In streptococcal general infection there is nearly always suppuration in the joints; in staphylococcal infection, suppuration in the bones. Lastly, infective emboli or propagated thrombo-phlebitic abscesses (metastatic) may occur in all the organs, especially in staphylococcal infection. In this way multiple abscesses may appear in the skin. Metastatic abscesses may be cold and painless, and often contain few bacteria. Deeply situated subfascial and intermuscular abscesses often escape observation. Lastly, small multiple or large abscesses may occur in the lungs, heart, liver, kidneys, etc. According to *Waldeyer*, these abscesses are due to plugging of the smallest vessels. For example, plugging of the central artery of the optic nerve causes panophthalmitis, while plugging of a terminal artery in the lung causes an infarct. In this advanced stage of general infection, there is often frequent bleeding from the wound at the seat of the local infection, due to affection of the arteries. In the pre-antiseptic period many cases of amputation were fatal owing to this so-called septic secondary hemorrhage. In this stage bacteria are nearly always found in the blood. While an increase of bacteria in the blood is a bad sign, their disappearance is not always a good sign for the further progress of the case; for after the disappearance of bacteria from the blood in many diseases, the action of their toxins (toxinæmia) becomes manifest. Streptococci are more easily demonstrated in the blood than staphylococci.

According as the onset is gradual or sudden, and according to the degree and the course of the general infection, a many-sided but unmistakable clinical picture is produced.

The chronic forms of general infection, which occur after long-standing fistulas, suppuration and necrosis, are characterized by their gradual development and by

the slight severity of the symptoms. Many cases, however, are fatal from heart failure or albuminuria; or the chronic form may become acute. In chronic general infection there are often long periods free from fever, followed by rigors and rise of temperature. In the chronic forms metastatic abscesses are more common. In such cases recovery may take place after removal of the primary cause, but it requires several months to restore the weakened body. Again, acute general infection may become chronic, and occasionally end in recovery.

It is only young and robust bodies that can offer an effective resistance against such a destructive morbid condition, and then only in the early stages of infection. The organism cannot withstand the destructive action of a fully developed general infection. These cases are all fatal. Even in the early stages of general infection the heart may become so weak by the action of toxins, that death occurs from collapse before the full development of the clinical picture. Staphylococcal infection, with its tendency to metastatic formation and its remittent type of fever, is more likely to recover than streptococcal infection; this is generally fatal in a few days, with continued fever and increase of all symptoms, but without metastatic formation.

Differential Diagnosis. Although the clinical symptoms of a typical case of general infection are unmistakable, cases in which the origin of infection remains unrecognized, or cases of chronic general infection may be mistaken for typhoid, miliary tuberculosis or acute rheumatism. Severe inflammations, erysipelas (especially hemorrhagic bullous erysipelas) may be associated with such high temperature and rigors, etc., that it is difficult at first to distinguish whether the symptoms are due to the local condition, or to the commencement of general infection. The progress of the disease will decide. It must, how-

ever, be borne in mind that in these cases there is generally already commencing general infection, especially in cases of progressive inflammation.

Treatment. Apart from cases in which the most acute form of general infection arises from comparatively slight lesions, some cases may be cured by proper treatment of infected wounds (Fig. 93), and by early diagnosis of commencing general infection. Special attention must be devoted to the place of entry of the infection. Free incisions are here required. Infected joints must be resected. In some cases of severe general infection and progressive suppuration in the extremities, amputation should not be too long delayed.

In threatening general infection from purulent thrombo-phlebitis of the large veins, ligation should be performed; for instance, of the jugular vein and anterior facial vein in carbuncle of the face; of the internal jugular vein in otitis media. Metastatic abscesses must be opened early. Metastatic joint effusions should be incised. Pleural effusions require aspiration or resection of the ribs. Suppuration in the internal organs (liver, brain, kidneys) require operative interference.

Antipyretics are best avoided on the whole, as they obscure the symptoms and weaken the heart. In severe cases high temperature may be reduced by tepid sponging. The heart must be supported by stimulants. Nourishing diet is required (if necessary by esophageal tube). Subcutaneous or intravenous injections of saline solution are often useful. Subcutaneous injections of nucleinic acid with salt solution have been recommended. Not more than two hundred to three hundred cubic centimeters of solution should be injected at one time into the veins. Injections of colloidal silver, anti-streptococcal serum and polyvalent serum (*Aronsohn*) have generally no influence on the disease.

On account of the danger of infection to others, the patients should be isolated and treated by special attendants. The body must be frequently bathed with alcohol or spirit of camphor to prevent the formation of bedsores, especially on the back and buttocks. The wound at the seat of infection must be dressed at least once or twice a day to prevent accumulation of pus. Frequently changed moist dressings are the best. Iodoform gauze should be avoided, as it is rapidly decomposed by the secretion and gives rise to toxic symptoms. Disinfection of the wound with strong antiseptics is to be avoided on account of its injurious effect on the tissues. The affected parts of the body should be completely immobilized. Rubber gloves should be worn when dressing the wounds, and no aseptic operation should be performed on the same day by the surgeon who dresses them.

After recovery from general infection great care must be taken of the body, in order to give encapsuled traces of the disease an opportunity to heal. These encapsuled metastatic foci may at any time (even after some years) become virulent from some exciting cause, and give rise to fresh infection. Patients often succumb, after some years, to nephritis, endocarditis, pleurisy or pneumonia. In these cases strychnine is useful.

Fig. 108 shows a case of acute general infection arising from a subcutaneous whitlow, which was insufficiently incised and extended to the tendon-sheath and the joint. The temperature rose to 41° C. (106° F.), with rigors; remained high for a few days and then became remittent, during the formation of several subcutaneous metastatic abscesses. An abscess developed gradually in the thigh; this was incised, and thin pus containing a few staphylococci evacuated. Staphylococci were also present in the blood for some time. Other symptoms were—dry tongue, jaundice, slight delirium, and diarrhea.

The wound in the finger was dry and unhealthy. After disarticulation of the finger there was no extension of infection to the hand, and the whole condition improved. Under the above-mentioned treatment, with injection of saline solution, etc., recovery took place in a few months. Several metastatic abscesses required incision during the course of the disease. After removal of the finger, bacteria were no longer found in the blood—a proof that the virulent bacteria in the blood were derived from the seat of infection. The pulse remained rapid for a long time after recovery.

GASPHLEGMONE (*Gaseous phlegmon*)
ŒDEMA MALIGNUM (*Malignant Œdema*)
PHLEGMONE EMPHYSEMATOSA—GANGRAENOSA
(*Gangrenous, emphysematous phlegmon*)
Plate LXXXVII, Fig. 109.

We have already mentioned (Fig. 101) the progressive putrefactive inflammation which often occurs in necrosed tissues, and in the wounds of diabetics. Similar conditions of progressive inflammation, under various names, accompanied by rapid necrosis and the formation of gases in the tissues, give rise to general infection, and run an unfavorable course.

Pirogoff described these cases as acute purulent œdema, *Maisonneuve* as fulminating gangrene, others as gasphlegmon, gangrenous phlegmon, etc. The putrid necrosis of wounds known as "hospital gangrene," which was so common in the pre-antiseptic days, appears to be nothing more than putrefactive inflammation due to gas-forming bacteria. All these conditions are best included under the name *progressive gaseous phlegmon*. The causes of these phlegmons are not well known, as they are anærobic bacteria which have not yet been well differentiated from each other by bacteriological methods. They are found most often in dust, manure and putrid flesh.

The bacillus of malignant œdema, the *bacillus emphysematosus* and the *proteus vulgaris* are the bacteria at present found, generally in symbiosis with the ordinary pus-forming bacteria, especially streptococci. By this symbiosis the growth of the anærobic bacteria is at first made possible in open wounds, and through the combined action of both

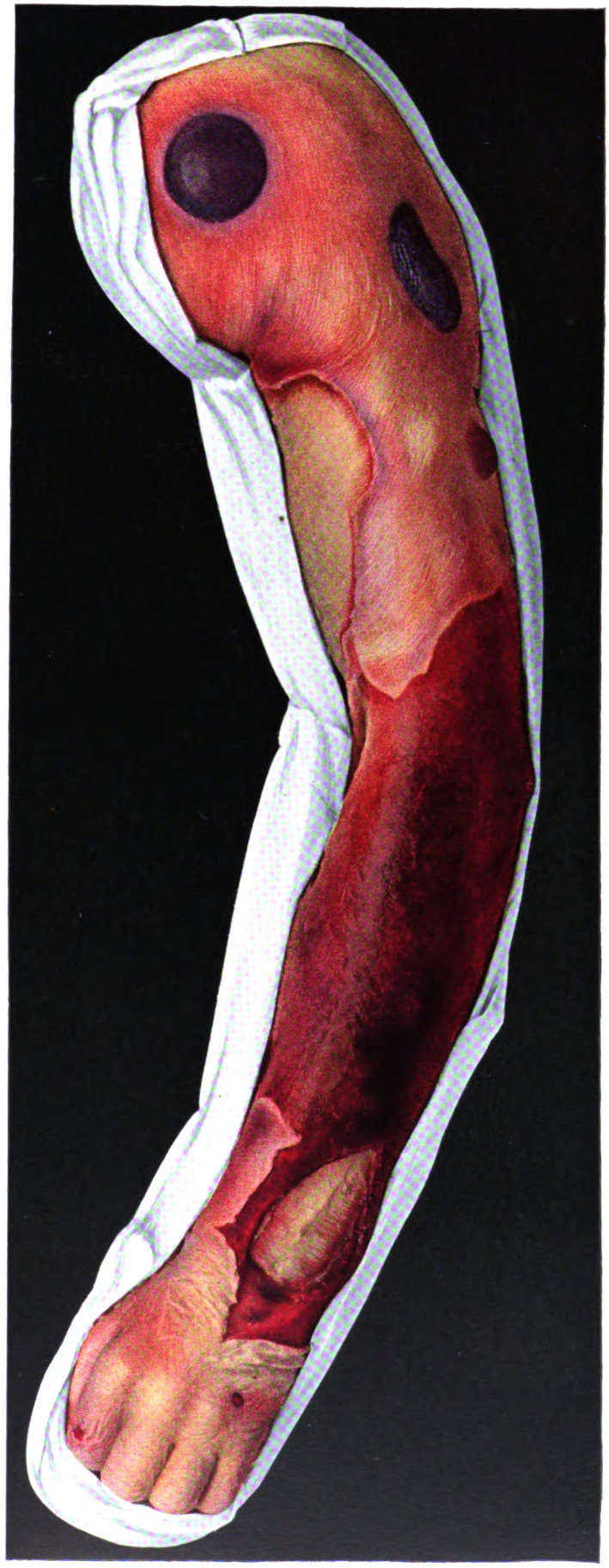

Fig. 109. Oedema malignum. Phlegmone emphysematosa-gangraenosa.

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forms of bacteria rapid and extensive destruction of tissue may be caused. Sometimes gaseous phlegmon is found after quite harmless lesions of the skin (Fig. 109), also after compound complicated fractures with small wounds.

Gaseous phlegmons occur in the extremities; on the back, in connection with bedsores; in operative wounds on the rectum, through infection by fæces; in the penis, scrotum and perineum, from lesions of the urethra with extravasation of urine; in the neck, after lesions of the esophagus and pharynx. The progress of gaseous phlegmon is extremely rapid; in a few hours large portions of the body are affected by the rapid formation of gas. As gaseous phlegmon may occur after apparently slight injuries, it is necessary to emphasize the necessity of frequent dressings in order to control the progress of infection.

The wound becomes dry, coated and fetid, and extensive swelling rapidly extends from it on all sides. The discharge from the wound is brownish or greenish, fetid, and mixed with necrotic shreds of tissue. High temperature, rigors, severe pain, anxiety and later on delirium and frequent pulse indicate the onset of general infection.

The circulation is obstructed by the great pressure of gas in the tissues. The skin of the extremities becomes pale and cold, and presents brown and green spots, and punctiform hemorrhages. Small vesicles filled with dark fluid then appear, which later on become larger; finally the whole epidermis of the affected parts is raised, and underneath it is offensive, dirty fluid. In other places the skin is reddish brown, hard, and infiltrated. There is no formation of a circumscribed fluctuating collection of fluid, but the tissues are saturated with fetid, sanious fluid containing bubbles of gas. On pressure the characteristic crepitation of cutaneous emphysema is heard. The infiltration is seen best after incision. The tissues cannot be distinguished from each other—muscles,

fascia and periosteum are transformed into sodden, homogenous, greenish shreds. If the medullary cavity of a bone is opened, it is filled with sanious fluid. Sometimes circumscribed cavities containing fluid and gas are found under the skin. Pressure of gas may cause gangrene of the peripheral parts of the extremities, resembling the putrefaction of a corpse (Fig. 109). At the same time there is rapidly extending lymphangitis, in the form of reddish-blue or reddish-brown cords; the color being due to congestion in the tissues. The lymphatic glands are infiltrated and painful. The veins are affected with thrombo-phlebitis. Finally, the arteries are destroyed, and severe hemorrhage ensues. The neighboring joints are filled with sanious fluid (*e.g.* the hip joint after extravasation of urine.

The formation of gases in the subcutaneous tissue may extend to large portions of the body; for instance, from the neck to the thorax and abdomen, and from the coccyx over the whole of the back. Death generally occurs from general infection, when the formation of gases is found in the internal organs at the autopsy. Gaseous phlegmon in the neck may cause death from œdema of the glottis or from mediastinitis. In spite of the general infection bacteria are not usually found in the blood.

Differential Diagnosis. Gaseous phlegmon in the early stages may be mistaken for progressive streptococcal inflammation. Hemorrhagic bullous erysipelas (Fig. 91) and anthrax (Figs. 112 and 113) may also cause great swelling of the skin with formation of bullæ. However, gaseous phlegmon is distinguished from the above by its rapid course, by the necrosis of the tissues, by the fetid secretion containing gases, and by the crepitation in the œdematous parts. In doubtful cases bacteriological examination must be made.

Treatment. Early and free incisions are indicated to open up the tissues and deprive the anærobic bacteria of their conditions for existence. In compound fractures with infection of the bones and joints, amputation is necessary to save life. If the gaseous infiltration has already extended above the seat of fracture, amputation may be performed a short distance above this point, and the infiltrated tissues of the stump freely incised. The wound should be dressed with dry aseptic tampons (*not* iodoform), or moist dressings with mild antiseptic lotions. Disinfection with strong lotions is injurious.

In extravasation of urine external urethrotomy is required, besides free incisions. In gaseous phlegmon of the neck a preliminary tracheotomy is necessary before making incisions, on account of the danger of œdema of the glottis.

Fig. 109 shows a characteristic case of gaseous phlegmon. In a young man two small abrasions were caused by a meat-knife, one on the index finger and one over the fifth metacarpo-phalangeal joint. In a few hours the forearm became enormously swollen, and in a few days the swelling extended over the whole arm. The patient became delirious and finally completely comatose. After incision, the tissues were found infiltrated with fetid sanious fluid containing numerous necrotic shreds. The elbow and shoulder joints were full of sanious fluid. The fingers were cold. Bacteriological examination showed the presence of putrefactive bacteria and streptococci. There were no bacteria in the blood. There were the usual signs of severe general infection (dry tongue, jaundice, etc.). In spite of free incisions, and disarticulation at the shoulder joint on the third day, the patient died.

LYMPHADENITIS (BUBO) INGUINALIS DIFFUSA

(*Diffuse Inguinal Adenitis* (*Bubo*)

Plate LXXXVIII, Fig. 110.

Pyogenic affection of the lymphatic glands has already been mentioned in the case of glandular inflammation in the neck (Fig. 102). The lymphatic glands act as barriers which stop the bacteria brought to them by the lymphatic vessels and destroy them, unless they are too numerous and virulent, when they become themselves affected. Besides the common pyogenic affections of the glands of the neck, the axillary and inguinal glands are often affected. The inflammation may be acute or chronic. Injuries, eczema, and pyogenic affections such as whitlow, abscess, lymphangitis or erysipelas may give rise to an acute purulent lymphadenitis or to chronic lymphadenitis, usually staphylococcal. The point of origin is often invisible, for a small excoriation of the skin may heal before the lymphangitis to which it gives rise becomes apparent.

Abscesses of unknown origin (*e.g.*, in the abdominal wall) generally arise from suppurating aberrant lymphatic glands. The inguinal glands (inguinal bubo) may be affected after ingrowing toenails, excoriations (Fig. 110), soft chancre or gonorrhea. In the last case gonococci are found in the pus.

The acute forms are very painful and prevent movement of the limb. The skin becomes red, and is at first movable over the inflamed glands; but it gradually becomes infiltrated and bluish red in color. Pyogenic infection of the lymphatic glands may give rise to diffuse suppuration of the surrounding tissue (periadenitis) which may extend rapidly in the sub-

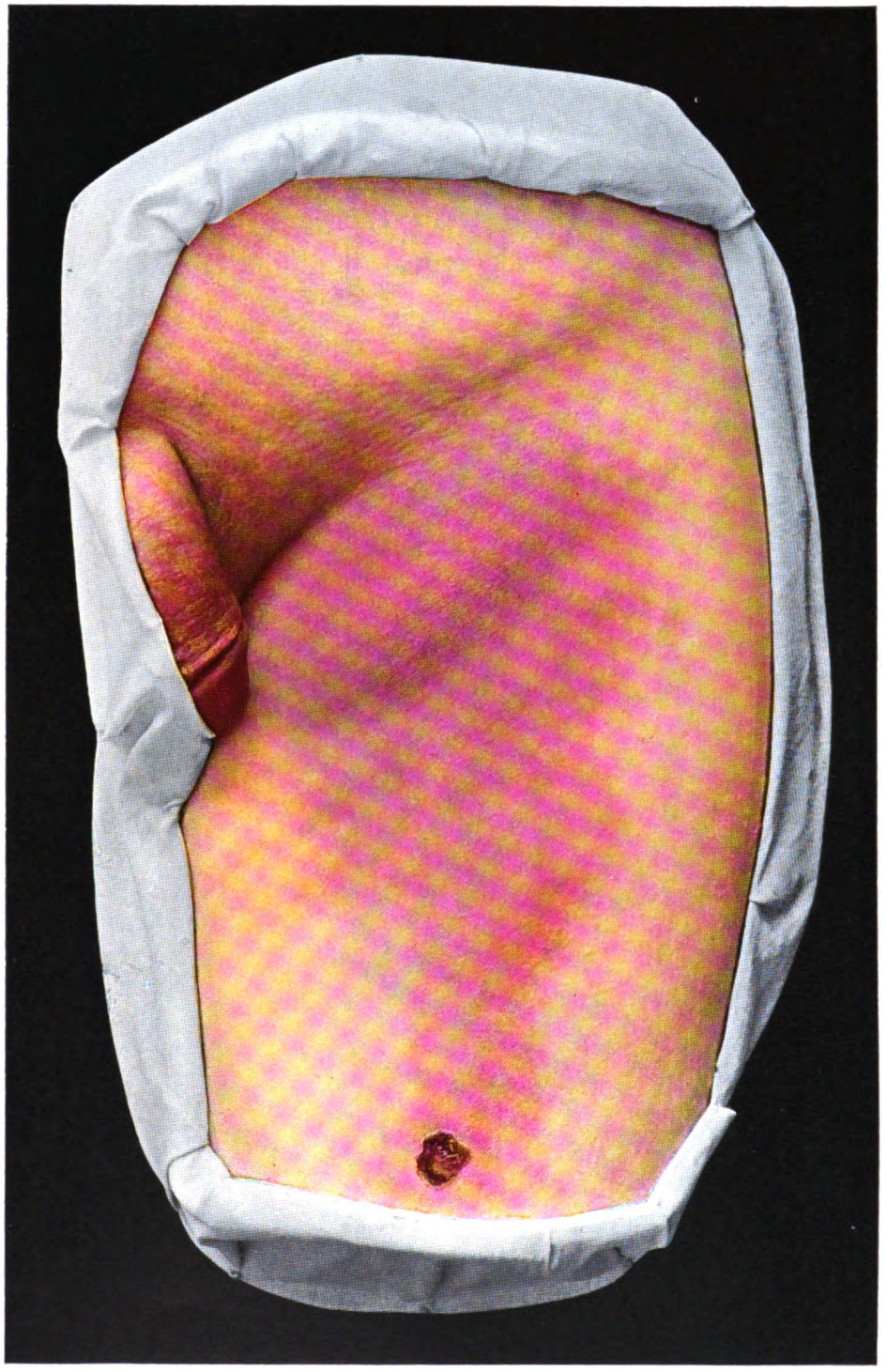

Fig. 110. Lymphadenitis inguinalis diffusa (Bubo).

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cutaneous tissue, both superficially and deeply (Fig. 110). In this form there are rigors, fever and constitutional disturbance. More often the inflammation is localized and gives rise to a circumscribed abscess (Fig. 114). The skin becomes thin and the pus is discharged through a fistula. After this the pain subsides; but the fistula does not heal, because the whole gland is generally necrotic and is gradually cast off, giving rise to infection of the neighboring lymphatic glands and the formation of multiple fistulas.

Diffuse suppurative lymphadenitis causes still greater destruction for there is not only necrosis of the glands themselves but also of the periglandular tissue, and even of the subcutaneous tissue in extensive cases. Moreover, burrowing abscesses may develop in remote places; for instance, in the pelvis after inguinal adenitis, and in the retro-pharyngeal tissue after cervical abscesses. Again, general infection may occur from thrombo-phlebitis (*e.g.*, from thrombo-phlebitis of the pelvic veins after inguinal bubo. All these complications can be avoided by early incision.

In the chronic forms inflammatory symptoms are absent. A slightly painful thickening develops in one or more glands, after long-continued irritations, inflammation in the neighboring parts, eczema, pediculosis, ulcers, etc. Finally, a small, irregular, movable swelling is formed in the subcutaneous tissue, covered by normal skin. Recovery takes place after removal of the cause; but in long-standing cases a permanent swelling may remain (fibrous hyperplasia).

Differential Diagnosis. Acute lymphadenitis is characteristic and easy to diagnose by occurring in the situation of the various groups of lymphatic glands. The diagnosis of submaxillary and cervical lymphangitis from alveolar periostitis, dermoids, sebaceous cysts and tuberculous abscesses has al-

ready been given (Fig. 102). Acute lymphangitis may be mistaken for sweat-gland abscesses, especially in the axilla, but these are usually small, multiple and circumscribed. In the inguinal region a hernia may be mistaken for a bubo, especially when the sac of the hernia is inflamed and is situated over the glands. This error is more likely to occur in incomplete hernias in women. In these cases diagnosis is often only made after incision. Suppuration arising from neighboring bones or joints may also simulate lymphadenitis. Tuberculous lymphangitis may cause inflammatory infiltration, and painful enlargement of the glands accompanied by fever; but the glands are softer and of less uniform consistence. If a fistula is present the diagnosis is more easy (Fig. 125). Tuberculous abscess is of slower development, and is generally associated with various degrees of infiltration of neighboring glands. Lastly, the thin greenish pus is characteristic.

Chronic lymphadenitis may be mistaken for metastatic carcinomatous disease of the glands; *e.g.*, of the inguinal glands after cancer of the anus. These glands, however, are hard and more or less fixed. Syphilis gives rise to multiple hard infiltrations of the lymphatic glands in various parts of the body.

Treatment. Pyogenic infection of the lymphatic glands can often be avoided by removal of the primary cause. In acute lymphadenitis early incision will prevent the complications mentioned above. If this is neglected, not only the whole of the lymphatic glands of the affected region, but also the subcutaneous tissue may undergo necrosis; also œdema or elephantiasis of the extremity may develop, owing to the obstruction of the lymphatic circulation. Elephantiasis may also occur after total extirpation of the lymphatic glands (Fig. 71). This may be avoided by taking care not to remove too much of the fatty connective tissue along with the glands; this tissue

carries on the lymphatic circulation after removal of the glands, and new glands are also formed from it. In the groin and axilla a careful dissection of the glands must be made, avoiding the great vessels.

Circumscribed abscesses are best opened by a free incision. Treatment by poultices or icebags, aspiration, puncture and injection of various fluids, massage and inunction of mercurial ointment are best avoided. The affected parts should be immobilized to prevent extension of the infective process. Patients should, therefore, stay in bed. Commencing infection of the lymphatic glands often undergoes spontaneous resolution. Acute lymphadenitis of the neck caused by infections such as diphtheria, may subside spontaneously; so may chronic lymphangitis when it is not of too long standing, and when the cause is removed. Inunction with iodide of potassium or iodine-vasogen ointments is useful in chronic lymphangitis. In cases of large glands causing pain, or of multiple fistulas connected with chronic lymphadenitis, the glands should be extirpated, and the wounds plugged for a long time with iodoform gauze to prevent relapse.

Fig. 110 shows a case of acute lymphadenitis of the inguinal region, occurring after an excoriation of the skin of the thigh, which has already scabbed over. Infiltration of the skin and subcutaneous tissue extends from the genito-crural fold down the thigh. The symptoms were pain and difficulty in walking, followed by fever and rigors. Under an anæsthetic an incision was made below and parallel to *Poupart's* ligament. The inguinal glands were swollen, and contained numerous foci of suppuration; but there was no extensive necrosis, nor any large collection of pus. The wound was plugged with iodoform gauze and the leg immobilized on a splint. The wound healed after a part of the gland which had necrosed came away.

ARTHRITIS GONORRHOICA PHLEGMONOSA

(*Phlegmonous Gonorrheal Arthritis*)

Plate LXXXIX, Fig. 111.

In the course of both acute and chronic gonorrhea the joints may be affected by general gonococcal infection of the blood. In the acute stage of gonorrhea, arthritis may be caused by the passage of bougies or by overexertion, etc. In women it may occur during pregnancy. In chronic gonorrhea it may be caused by sexual excess. Gonococci may remain for a long time in a latent state encapsuled in the mucous membrane, and when set free by mechanical irritation may again become virulent. Recurrence of gonorrheal arthritis may take place in cases of neglected gonorrhea, also after a fresh attack of gonorrhea.

Through invasion of the joints by the gonococci and their toxins inflammation is set up which may be serous, fibrinous or purulent. Most commonly the arthritis is fibrinous, suppurative arthritis being rare and generally caused by mixed infection. One or several joints may be affected at the same time, or successively. Acute gonorrheal arthritis is very sudden in its onset, and characterized by severe pain, preventing any movement of the affected joint. In a few hours the soft parts become infiltrated and œdematous, the infiltration remaining more or less limited to the region of the joint, or spreading to the neighboring muscles and tendons. The skin is red and tense (Fig. 111). In severe cases there is high fever and complete loss of function. In chronic gonorrheal arthritis there are usually aching pains in the joint before the arthritis becomes evident.

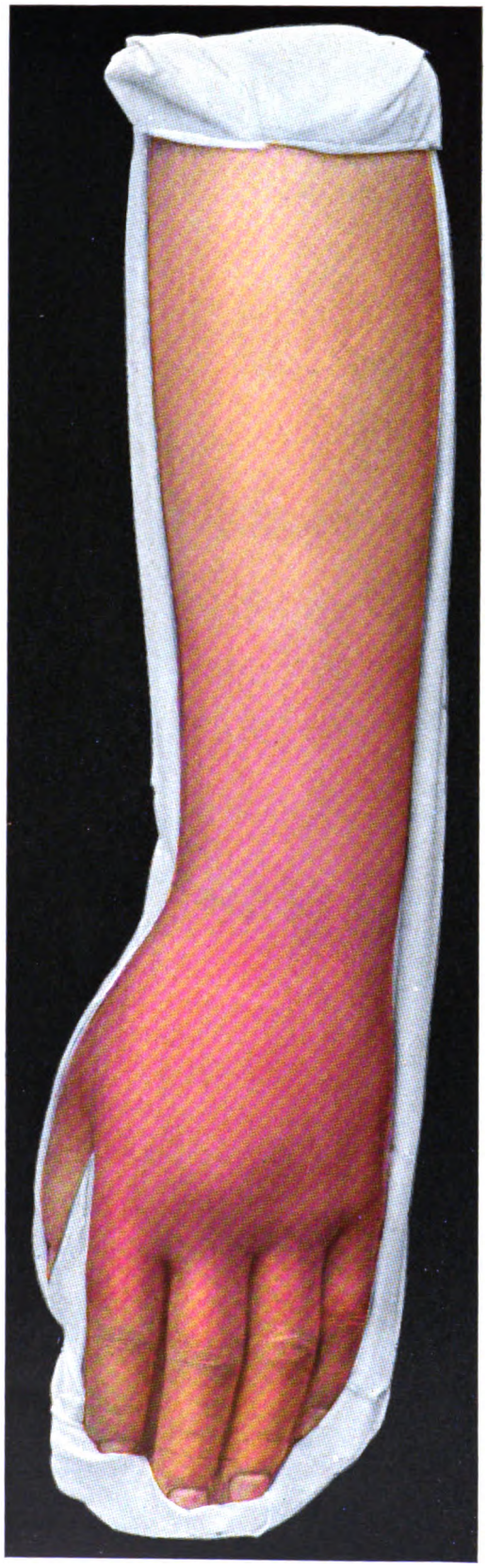

Fig. 111. Arthritis gonorrhoica phlegmonosa.

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The knee joint is most often affected in men; the elbow and wrist in women. The hip, ankle and temporo-maxillary joints are also often affected.

In cases of serous or sero-fibrinous effusion, limited to the joint, the swelling generally subsides in one or two weeks, and recovery takes place without loss of function. In the more common form of *fibrinous arthritis*, however, the process is more severe, especially when the infiltration extends to the periarticular tissue and the soft parts. In these cases the arthritis is accompanied by fever and rigors, and there is sero-fibrinous effusion into the neighboring parts, but no formation of pus. The inflammation affects not only the synovial membrane, but may cause destruction of the cartilage and extend to the bone. This may result in fibrous, cartilaginous or bony anchylosis (X-ray examination). Destruction of the capsule of the joint may cause subluxations or dislocations, and the prolonged immobility may lead to muscular atrophy.

In the rarer forms of suppurative arthritis there is continued high fever with rigors, and severe constitutional disturbance. The skin is red and there is great swelling of the affected parts.

If several joints are affected different forms of gonorrheal arthritis may occur in the various joints. Multiple relapsing arthritis may reduce the patients to a deplorable condition, as they often cannot walk or use their arms. Such cases may be fatal from gradual exhaustion.

Differential Diagnosis. Gonorrheal arthritis may be mistaken for acute rheumatism; but the latter usually affects a greater number of joints, and the acute stage of inflammation is not so prolonged as in gonorrheal arthritis. Purulent gonorrheal arthritis must be diagnosed from other suppurations in joints by the history, by the presence or history of acute or chronic gonorrhea, or by bacteriological examination after puncture of the joint.

Chronic gonorrheal arthritis is often difficult to distinguish from certain forms of syphilitic arthritis, especially when both diseases have been contracted together.

Tuberculous arthritis is usually easy to distinguish by its characteristic signs (Fig. 125).

Treatment. On account of the severity of the disease and the possibility of a fatal ending, especially from endocarditis, the prophylactic treatment of gonorrhea is important. Washing immediately after coitus, vaginal injections in women, the instillation of a few drops of weak silver nitrate solution into the urethra after coitus, and the avoidance of any kind of irritation (alcohol, etc.), will often prevent gonorrheal infection. Gonorrhea should be regarded as a serious disease and treated accordingly. In gonorrheal arthritis the urethra should always be examined, and treated if gonorrheal urethritis is present.

The subacute or serous forms of gonorrheal arthritis subside in one or two weeks after rest in bed; but too early movement may cause relapse. In fibrinous arthritis, on the other hand, too long immobilization may lead to anchylosis. Immobilization (by plaster of Paris bandages, or better by extension splints) should, therefore, not be continued longer than one or two weeks; after which gentle massage, active and passive movements or hot air treatment should be tried. *Langenbeck* has recommended "animal baths" for cases of stiffness; *i.e.*, placing the affected part in the viscera of a freshly killed animal, to obtain the effect of animal heat. Sandbaths are also worth a trial. If an acute relapse occurs in the course of the disease the joint must be again immobilized. In any case, movements of the joint must be carried out after two or three weeks; otherwise bony anchylosis may occur. Injections of morphine may be given before the performance of massage or passive movements, or cocaine may be injected into the joint (0.05 cubic

centimeters of a five per cent. solution). Injections of carbolic acid and protargol solution into the joint have also been recommended. The best method of treatment would be injection of antitoxin, as the inflammation is primarily caused by the gonotoxin.

[In one case a good result was obtained by injection of meningococcus-serum *Bockenheimer.*]

By careful after-treatment complete function can generally be restored even in severe forms of gonorrheal arthritis.

Bier's treatment by passive hyperæmia has a good effect in these cases, and may be tried in all cases of gonorrheal arthritis where there is no suppuration. After application of the elastic bandages, the joints become painless (in about fifty per cent. of cases), so that the patients do not hold them so stiffly, and early movements can be performed, thus giving a better functional result. In severe cases the joints should be bandaged to protect them against injury, the bandages being frequently removed and movements performed. The elastic compression bandages should be applied at first for two or three hours, later on for twenty hours. This treatment may be carried out without danger in out-patient practice (polyclinic).

If there is much destruction of the joint, with subluxation or anchylosis in a faulty position, resection may be required. Fibrous contractures are common after gonorrheal arthritis; these can be corrected under an anæsthetic, and that function restored by appropriate after-treatment. In suppurative arthritis, which is often complicated by lymphangitis, lymphadenitis and other pyogenic conditions, arthrotomy or resection of the joint must be performed to avoid general infection. These cases require longer immobilization of the joint.

Fig. 111 shows a case of acute and painful swelling in the region of the wrist joint in a woman. The skin was red and tense. The swelling rapidly extended

to the forearm and to the fingers, so that the patient could not use the arm. The wrist joint and the metacarpo-phalangeal joints could not be moved. Examination of the genitals showed gonorrhea. On the following day the joint effusion increased and was partly evacuated by puncture. Gonococci were found in the fluid. Under treatment by passive hyperæmia the pain subsided in a few days and the acute inflammation became chronic. Massage, active and passive movements, combined with passive hyperæmia, restored the function in four weeks.

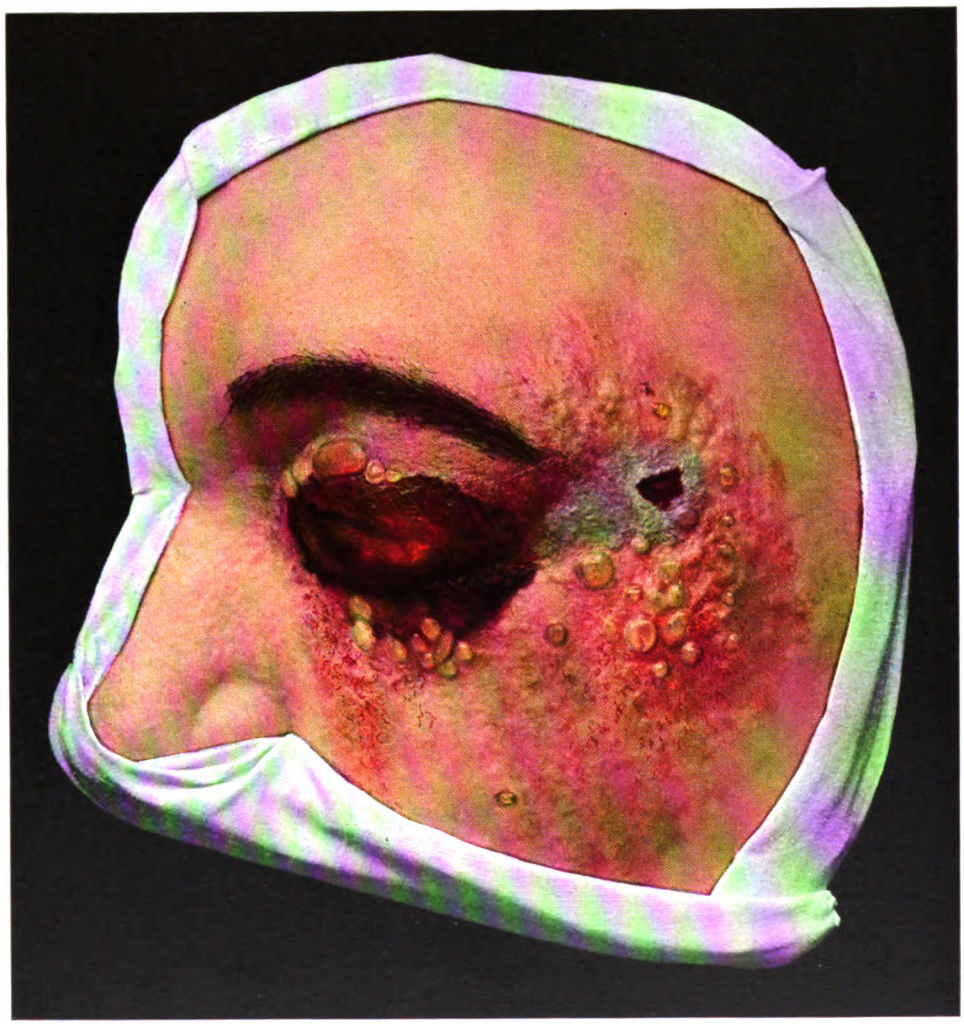

Fig. 112. Anthrax — Pustula maligna.

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ANTHRAX — PUSTULA MALIGNA

(*Anthrax—Malignant Pustule*)

Plate XC, Fig. 112.

ANTHRAX — NECROSIS (*Anthrax necrosis*)

Plate XCI, Fig. 113.

Anthrax (splenic fever) is a bacterial disease which occurs externally on the skin, and internally in the lungs and alimentary canal. The bacteria have a characteristic appearance. They consist of immobile rods (bacilli) with sharp, angular corners, and are often arranged in a row in long chains. In the center of the rods are clear spaces corresponding to spores, which are very resistant to dryness and heat. The anthrax bacillus was carefully studied by *Koch*, while *Pasteur* originated the protective inoculation of animals with attenuated cultures. The bacilli and spores are found in the alimentary canal of animals (horses and cattle); also in damp soil on which these animals graze, and in the skin, fur and excrements of animals infected with splenic fever (Rinderpest or cattle fever). Epidemics of anthrax are common in Egypt, as the excrements of animals are used as fuel for cooking purposes. The disease is common in Siberia in the skin trade and is known as Siberian plague. Butchers, skinners, ragsorters, tanners, paper makers and workers in horsehair are liable to anthrax infection. The disease has been observed in farmers, owing to the custom of treating horses and cows affected with colic by passing the hand into the rectum. The disease may also be transmitted by earthworms and flies. The bacilli may remain localized at the seat of infection, or may enter the bloodstream and give rise to metastatic foci in other places; while their toxins play a subordinate part.

The external form of anthrax occurs on the skin of the neck or face after small abrasions of the skin, through which the bacilli enter. The infection may be conveyed to the mouth by the finger, and the spores may thus be inhaled or swallowed, and give rise to anthrax of the lung or intestines respectively.

Anthrax of the skin has a very characteristic appearance. A small, red spot first appears, with fever and often rigors; this develops into a small vesicle with yellowish or turbid fluid containing anthrax bacilli (malignant pustule). The pustule ruptures and is replaced by a scab. At the same time the surrounding skin becomes green—a sign of commencing necrosis. The early appearance of necrosis of the skin is characteristic of anthrax (Fig. 112). The tissues become infiltrated in the same way as in carbuncle (cf. Fig. 89), and œdema occurs where the skin is loosely attached to the subcutaneous tissue (*e.g.* eyelids). The redness of the skin extends rapidly and irregularly, resembling erysipelas. Other vesicles appear and rupture, after which there is extensive necrosis of the skin (Fig. 113).

In the extremities, along with the above symptoms, there is always lymphangitis and lymphadenitis, which may form abscesses by mixed infection. There is always considerable constitutional disturbance, with fever, rigors, headache and rapid pulse. Multiplication of the bacilli in the blood gives rise to symptoms of general infection—dry tongue, jaundice, diarrhea and swelling of the spleen. Death may result from collapse in a few days.

External anthrax has a more favorable prognosis than internal (mortality twenty-five per cent.), but anthrax of the face is very dangerous. The more marked are the local symptoms the more likely is general infection. Moreover, anthrax of the face may easily infect the mouth, and thereby cause infection of the lungs or alimentary canal. In the milder forms of general infection metastatic inflammations

are caused by emboli in the skin, lungs, alimentary canal and brain; giving rise to pleurisy and pneumonia, ulcers of the gut, peritonitis and meningitis, which are generally fatal. Primary internal anthrax may also cause secondary infection of the skin by metastatic deposits.

The usual form of internal anthrax is that affecting the intestine, caused by infection from the mouth; by bacilli conveyed by the finger, or by eating the flesh of infected animals. This gives rise to hemorrhagic ulceration of the small intestine, with a tendency to gangrene. About eighty per cent. of cases are fatal from peritonitis or general infection. Bacilli are found in the stools.

In the lungs anthrax is more rare and is caused primarily by inhalation of the spores. It occurs among manufacturers of paper and horsehair and among ragsorters, sometimes in an epidemic form. The patients are suddenly attacked with symptoms of pneumonia and high fever. The sputum is blood-stained and contains anthrax bacilli. About eighty-nine per cent. of these cases are fatal from pulmonary œdema and pleurisy.

Both external and internal anthrax may occur simultaneously, and the disease is then almost always fatal from general infection.

Differential Diagnosis. Pyogenic infections, such as virulent streptococcal or putrefactive inflammations (cf. Fig. 109), and hemorrhagic bullous erysipelas may cause the formation of bullæ on the skin, and may, therefore, be mistaken for anthrax; but these affections run a different course and do not lead so quickly to necrosis of the skin. Glanders also gives rise to the formation of bullæ and gangrenous ulceration, but the characteristic carbuncular infiltration of anthrax is absent. In doubtful cases anthrax bacilli must be looked for in the fluid of the bullæ. This is especially important, as the

treatment of anthrax differs from that of the above-mentioned affections.

Treatment. Prophylactic treatment consists in strict supervision of trades in which there is a danger of anthrax infection. Skins of animals should be disinfected, and workmen should be warned of the danger of infecting the mouth from handling skins, rags, horsehair, etc., especially during meals.

The less the local infection is irritated, the less is the danger of the bacilli entering the blood. For this reason both incisions and the thermo-cautery are contra-indicated, as they often cause extension of the infiltration or even general infection (*von Bergmann*). Scabs and necrosed tissue must, therefore, be left to separate spontaneously. The infected area should be dressed with ointment to prevent auto-infection of the patient. If the disease occurs in the extremities they must be fixed on splints. Abscesses in the lymphatic vessels and glands caused by mixed infection must be opened. Extensive necrosis of the skin sometimes necessitates a plastic operation (Fig. 113). As in other infective diseases, nourishing diet, stimulants, absolute rest and isolation are required. In severe cases the injection of *Sclavo's* serum in the region of the infected area is recommended.

Fig. 112 shows a case of external anthrax in a tanner, which developed after a slight abrasion of the skin. It began as a red papule, followed by several vesicles full of yellow fluid containing anthrax bacilli. At the same time there was erysipelatous reddening of the skin, carbuncular infiltration of the tissues, and œdema of the eyelids. The vesicle at the point of infection ruptured and was replaced by a scab, round which the skin gradually became gray and necrotic. Fever and rigors set in, and the disease spread to the eyelids. Fresh vesicles appeared, with further gangrene of the skin after their rupture. The

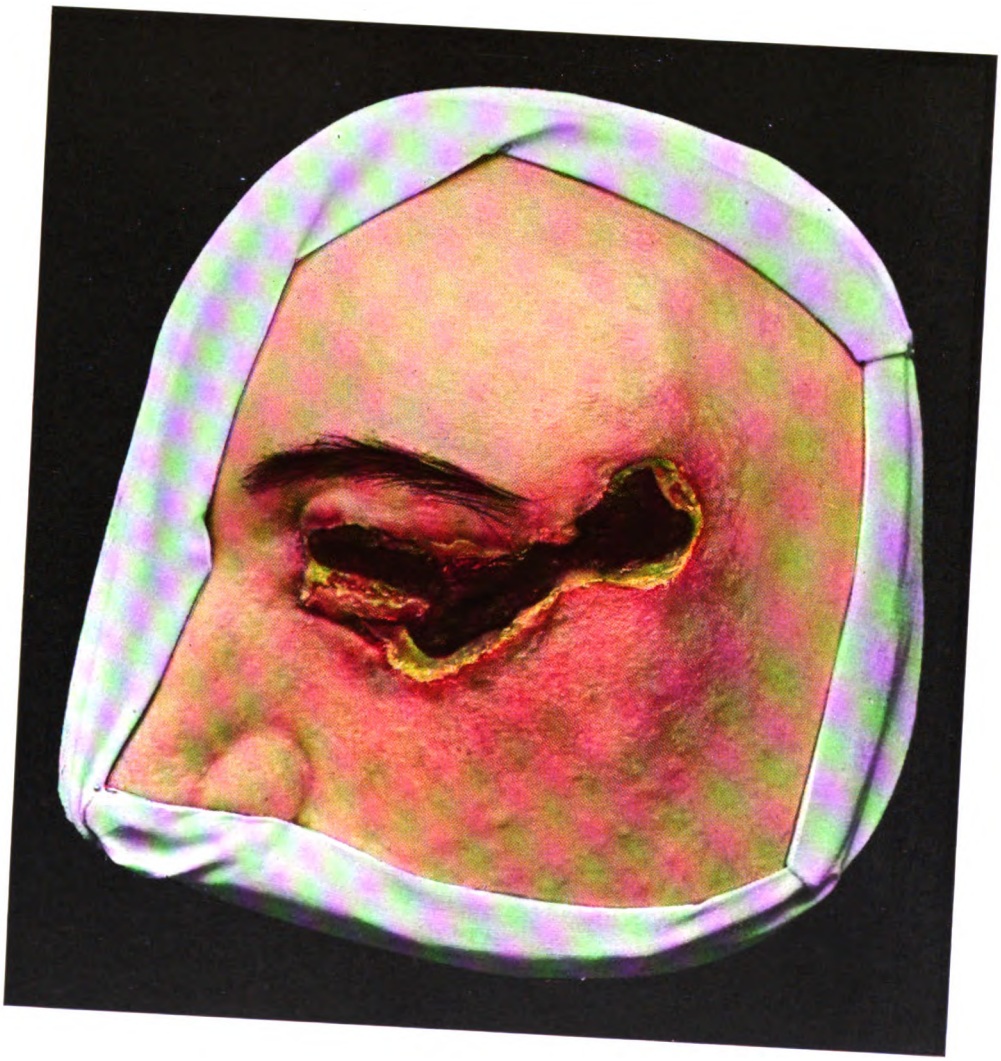

Fig. 113. Anthrax — Necrosis.

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affected area was covered with ointment and the symptoms gradually subsided, without internal anthrax or general infection supervening.

Fig. 113 shows the same case a few weeks after infection. The leathery, blackened, necrosed skin is separated by a zone of pus and slimy granulation tissue from the surrounding skin, which is still red and infiltrated. The necrosed skin is firmly adherent to the subjacent tissues. Removal of this by the knife or sharp spoon would only cause a further outbreak of infection. It was, therefore, allowed to separate gradually under treatment by moist dressings of peroxide and boric acid and ointments. In this case, after separation of the necrosed skin, the defect was repaired by a plastic operation, and the upper eyelid restored by a pedunculated flap. The patient recovered, in spite of the unfavorable prognosis in anthrax of the face and the severity of the local infection.

LYMPHADENITIS CIRCUMSCRIPTA ABSCEDENS

(*Circumscribed suppurative lymphadenitis*)

Plate XCII, Fig. 114.

In this case a circumscribed abscess formed in the lymphatic glands behind the ear, as the result of pediculosis of the scalp. The skin was red and thin at apex of the swelling. Fluctuation was present. There was no fever nor constitutional disturbance. The submaxillary lymphatic glands were enlarged and slightly painful on pressure. The abscess was incised, and the submaxillary glands inuncted with iodide of potassium ointment. Healing took place in a short time.

The figure shows the gluing together of the hairs and the punctiform deposits on them (nits) due to pediculosis. The frequent irritation has caused eczema of the scalp. Infection of the lymphatic glands is caused by infection through scratches. The treatment consists in removing the cause (*i.e.* the pediculosis) by rubbing in ten per cent. naphthol ointment. The nits can be removed by washing with soft, green soap, weak liquor potassæ or weak acetic acid and subsequent combing. The eczema generally disappears when the pediculosis is cured. The prophylactic treatment of pediculosis consists in cleanliness.

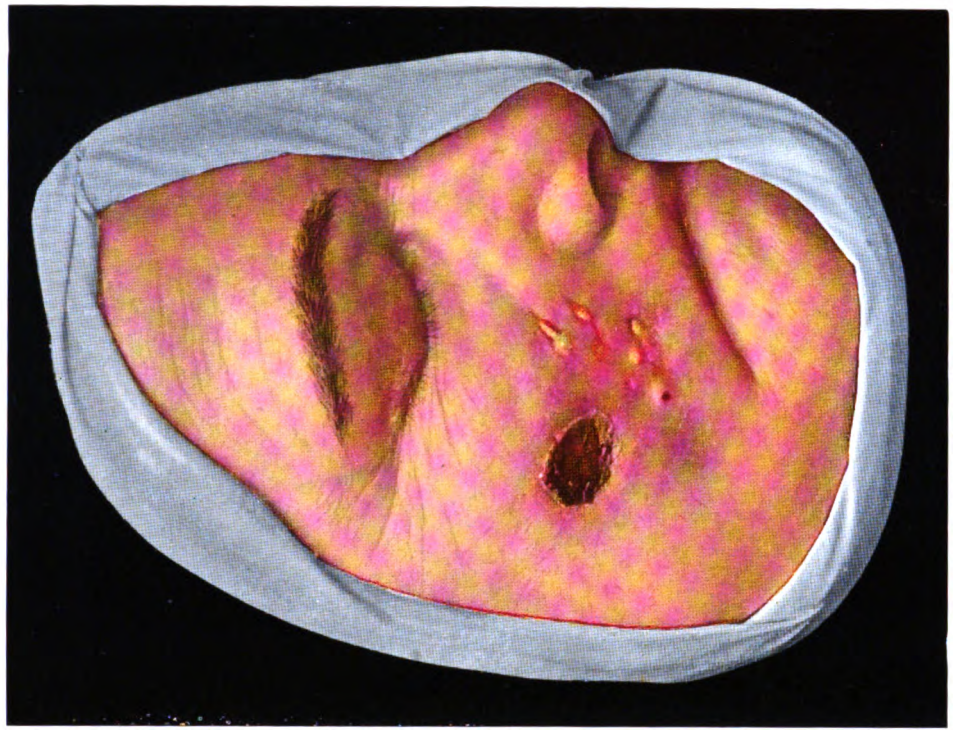

Fig. 115. Aktinomykosis incipiens.

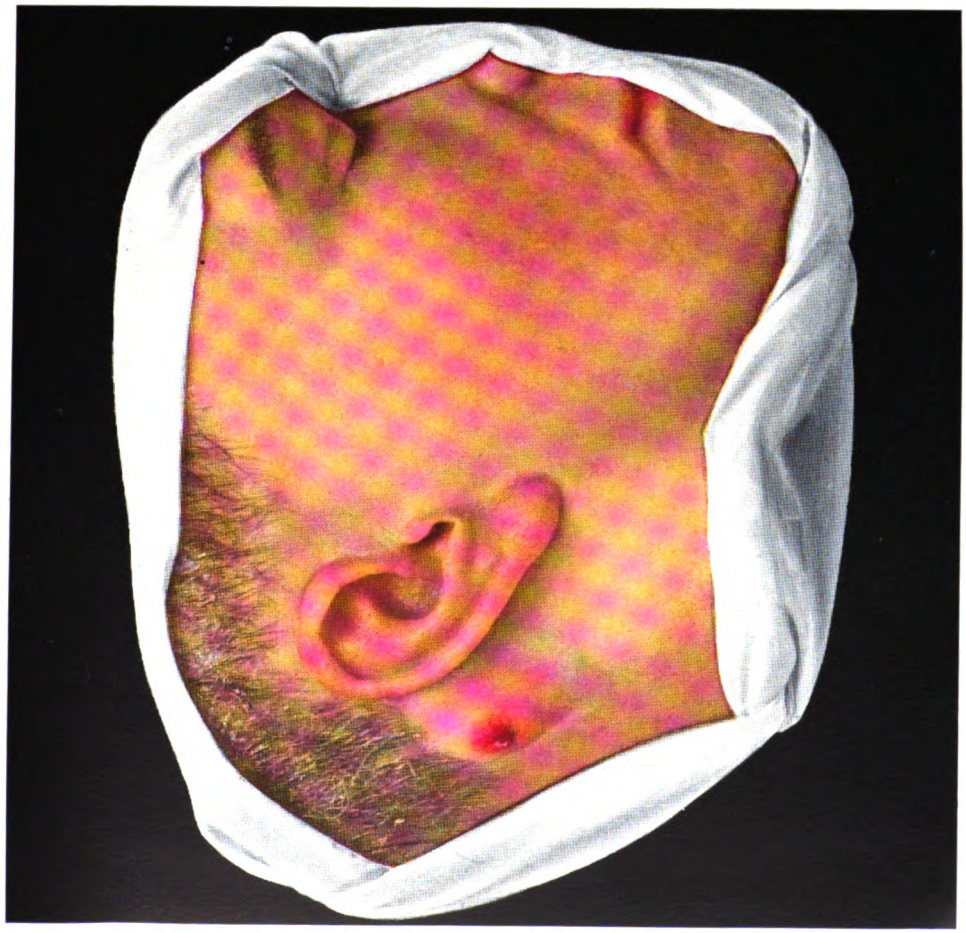

Fig. 114. Lymphadenitis circumscripta abscedens.

Actinomycosis

ACTINOMYCOSIS INCIPIENS (*Incipient Actinomycosis*)
Plate XCII, Fig. 115.
ACTINOMYCOSIS PROGRESSIVA (*Progressive Actinomycosis*)
Plate XCIII, Fig. 116.

Actinomycosis is a chronic infective disease caused by a fungus (*actinomyces*), which is called the ray-fungus on account of the radiating arrangement of its mycelium. Actinomycosis was first described by *Langenbeck*, and later by *Bollinger*, in the form of new growths in the lower jaw of cattle and horses. In 1878 *Israel* found yellow bodies in the pus from a patient who was supposed to have died of chronic pyæmia; the yellow bodies were found to be actinomyces. Later researches have shown that there are different forms of actinomyces. The fungus is best stained with *Levaditi's* silver nitrate method.

The fungus is found in corn, straw and flour. In countrymen who have the habit of chewing corn the mouth may become infected; either through a carious tooth, leading to infection of the bone, or through the parotid duct, leading to infection of the cheek. In the great majority of cases, therefore, we find actinomycosis in the mucous membrane of the cheek, the tongue, the jaw, the pharynx and the neck. It forms a stringy, nodular infiltration which, by becoming confluent, causes a swelling of wooden hardness. Acute inflammatory symptoms are absent. The skin becomes bluish red when the infiltration extends through the cheek or into the neck (Figs.

115 and 116). The infiltration extends gradually into the neighboring tissues, and by its unlimited progress resembles a malignant tumor. At the same time there is softening in the center of the infiltration with the formation of an abscess which discharges through several ramifying and anastomosing fistulas. The pus contains the characteristic yellow bodies, about the size of a pin's head. There is much induration around the fistulas which often prevents the discharge escaping. Granulation tissue is scanty, yellowish red in color, and rapidly disintegrated. The formation of abscesses is accompanied by a slight rise of temperature. Large abscesses may result from mixed infection; the yellow bodies are then often absent, the fungus being destroyed by the pus cocci.

In actinomycosis of the cheek a fistula is formed externally. If the infiltration is situated in the masticatory muscles there is trismus. The fungus may extend to the bones and give rise to enormous tumors. If the upper maxilla is invaded it may extend to the base of the skull and lead to meningitis or cerebral abscess. If the tongue is infiltrated it cannot be moved. When actinomycosis extends to the root of the tongue or to the pharynx there is difficulty in swallowing and later on in breathing. In these cases abscesses form which generally discharge through fistulas in the neck, and give rise to secondary actinomycosis of the skin. Primary actinomycosis of the skin has been observed through infection through lesions of the skin (*von Bergmann*).

The prognosis of actinomycosis of the buccal cavity is comparatively favorable, and by appropriate treatment two-thirds of the cases recover. On the other hand, by extension to the retropharyngeal tissue it may descend to the thorax or abdomen. Inspiration of secretion containing the fungus may infect the lungs. Invasion of the large veins of the neck resulting in metastatic foci has also been observed. From this circumstance the affection was formerly regarded

as chronic pyæmia especially as actinomycotic general infection gives rise to similar clinical appearances and eventually causes death by cachexia.

Actinomycosis of the lungs may occur from direct inspiration of substances carrying the fungus, besides infection from actinomycosis of the mouth. The prognosis is very bad. The symptoms are those of commencing phthisis. The lung becomes indurated and the pleura infiltrated, and abscesses discharge through the skin of the thorax. The disease may spread from the pleura to the pericardium, the vertebræ, the diaphragm and the abdominal cavity The patient becomes exhausted from empyema and multiple burrowing abscesses. The fistulas are difficult to follow owing to the hardness of their walls, so that relapses are common after incision, and the cases are usually fatal. Cases of recovery from actinomycosis of the lung have, however, been observed. Infiltration of wooden hardness between the ribs is always suggestive of actinomycosis.

The intestine may also be the seat of actinomycosis when material containing the fungus is swallowed. The ileo cæcal region is the part most often affected, in the form of hard tumor-like infiltration which may be so extensive as to prevent the passage of fæces. The disease may spread to the vertebræ, pelvic bones, abdominal organs, and may extend through the diaphragm to the thorax. There is often secondary actinomycosis of the skin. A fistula often forms near the umbilicus, discharging pus and sometimes fæces. The prognosis is somewhat more favorable than that of actinomycosis of the lung, but cases are often fatal from general infection.

When actinomycosis is visible externally the diagnosis is not usually difficult; the wooden infiltration, the multiple fistulas, the yellow granulations, and the yellow bodies mixed with the pus are characteristic. The diagnosis should always be confirmed by microscopic examination.

Differential Diagnosis. Actinomycosis of the cheek may, at first suggest lupus; but when the nodules have broken through, this mistake is no longer possible. Extensive infiltration of the cheek may be mistaken for tumors, especially when the jaw and the tongue are also affected; but the history of the formation of a cord extending often from a carious tooth, followed by swelling of the cheek will lead to the diagnosis. Actinomycosis of the tongue is distinguished from abscess or gumma by extending to the base of the tongue and causing immobility; also, in actinomycotic abscess the pus contains the characteristic yellow bodies. Actinomycosis of the neck may be mistaken for "wooden phlegmon," but the latter is generally unilateral and uniform, and does not form fistulas; actinomycotic infiltration extends round the whole neck, at first as a narrow zone, later on as several zones in the form of terraces one above another; the infiltration is also irregular. Actinomycosis of the lungs and pleura may be mistaken for tuberculosis, but the more advanced cases with fistulas are unmistakable. Actinomycosis of the intestine may be mistaken for tuberculosis or malignant growths, especially when it forms a tumor-like mass in the ileo-cæcal region.

Treatment. In extensive cases of antinomycosis of the buccal cavity attempts at total extirpation are useless, but healing may take place after free incision of abscesses and laying open all fistulas. Granulation tissue must be scraped away, and indurated tissue removed as far as possible. The incisions should be kept open for a long time by tampons. Carious teeth must be removed. In actinomycosis of the lung extensive resection of ribs is often necessary. In actinomycosis of the ileo-cæcal region resection of the gut may be necessary on account of intestinal obstruction or fistula. In other cases intestinal actinomycosis comes to the surface and then only requires

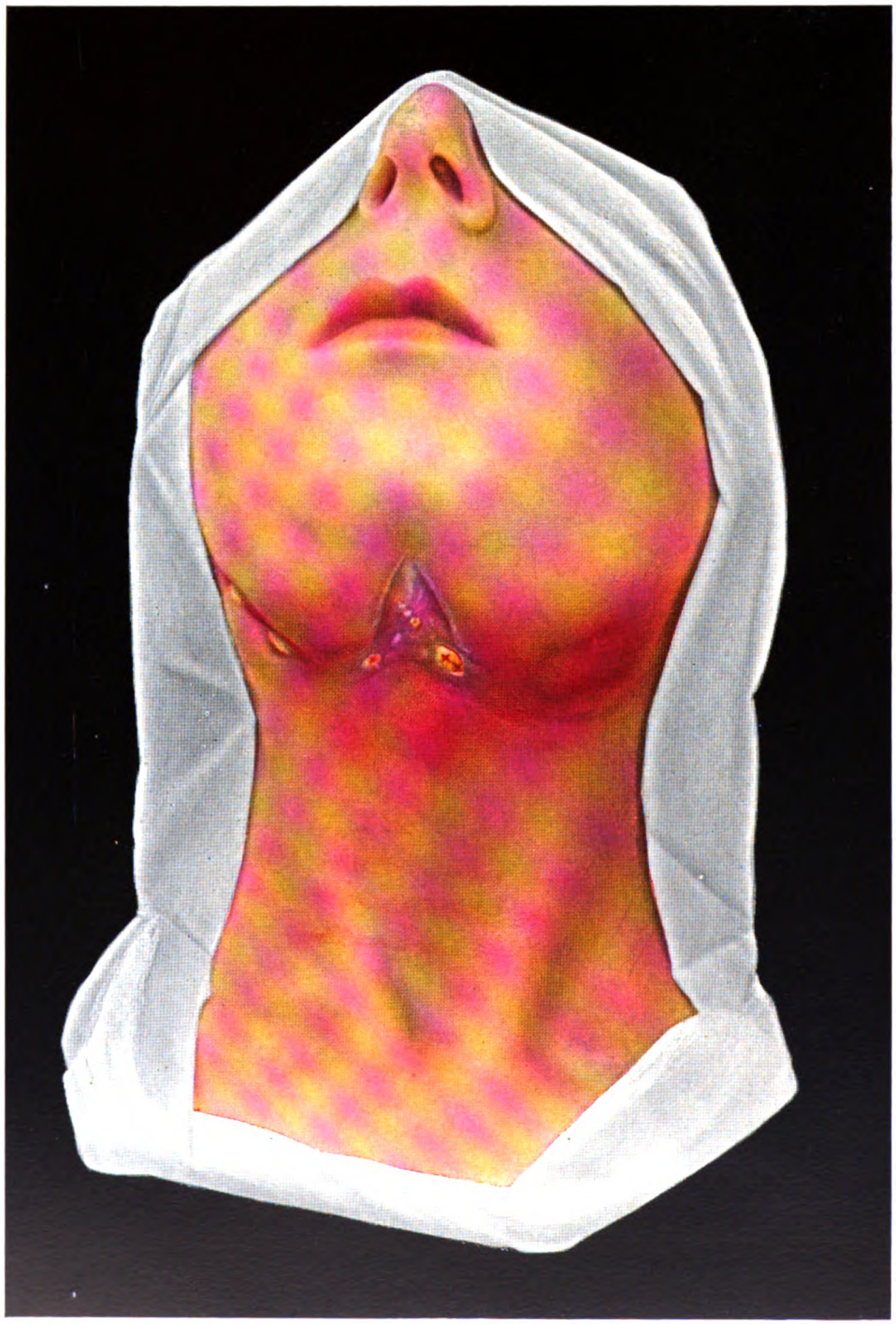

Fig. 116. Aktinomykosis progressiva.

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free incisions. Metastatic deposits in the bones (which can be detected by the X-rays) may require resection. General treatment consists in nourishing diet and the administration of iodide of potassium and arsenic.

Fig. 115 shows a case of actinomycosis of the cheek in an old countrywoman. Infection took place from a carious molar tooth. A cord-like growth extended from the root of the tooth to the gum, and thence to the mucous membrane and muscles of the cheek, giving rise to diffuse infiltration. The skin became bluish red and several small fistulas developed which discharged pus containing yellow bodies. The latter were found by microscopical examination to be actinomyces. A circumscribed patch of gangrene was caused over the malar bone by pressure of the infiltration. There was no fever and little trouble except a slight degree of trismus. Treatment by free incision and plugging.

Fig. 116 shows a case of extensive actinomycosis of the neck in a young countryman. The point of infection was not ascertained, and no changes were present in the mouth or pharynx. Hard, painless infiltration extended from one angle of the jaw to the other, finally spreading over the whole region of the neck. The skin was at first unaltered, but afterwards became dark red. A circumscribed abscess formed in the submaxillary region, which discharged pus mixed with yellow bodies through several fistulas, with yellow granulation tissue at their orifices. Both actinomyces and cocci were found in the pus, showing it to be a case of mixed infection.

The patient suffered from difficulty in breathing and in swallowing. Free incisions were made in the infiltration, the abscess was evacuated and the fistulas scraped.

LINGUA GEOGRAPHICA

(*Marginate Glossitis—Geographical Tongue*)

Plate XCIV, Fig. 117.

This affection is chiefly of interest on account of the possibility of its being mistaken for other affections of the tongue. The dorsal surface of the tongue is covered with segments of circles of a gray color, arranged irregularly and of various sizes. The intersection of these segments gives rise to an irregular polycyclic or "geographical" pattern. The condition is caused by patches of hyperkeratosis of the filiform papillæ which spread at the periphery and become normal in the center. The peripheral parts form the segments of circles and consist of an accumulation of desquamated epithelium. The condition occurs most commonly in infants, but also in young adults. It runs a benign course, and its cause is unknown. It has been attributed to a syphilitic origin by *Kaposi*, but this is doubtful.

Differential Diagnosis. Marginate glossitis must not be mistaken for leucoplakia. The two conditions have entirely different appearances. (Cf. Fig. 9.)

Treatment. No special treatment is required beyond mouth washes, painting with tincture of myrrh and avoidance of spicy foods.

Fig. 117 shows a case of marginate glossitis affecting the anterior two-thirds of the tongue. The whole tongue is divided into a series of projecting areas of a yellowish-white color. Between these areas are the gray segments filled with the secretions of the mouth. At the back of the tongue the surface is normal.

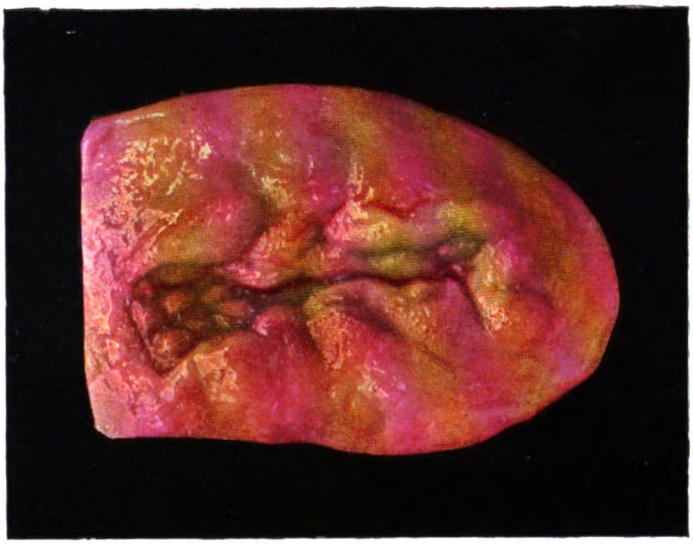

Fig. 119. Gumma linguae — Lingua bifida.

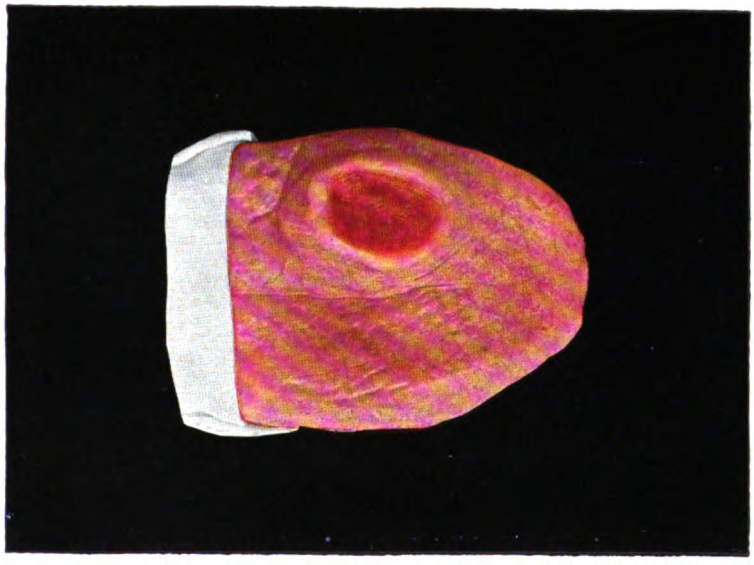

Fig. 118. Sklerosis syphilitica linguae.

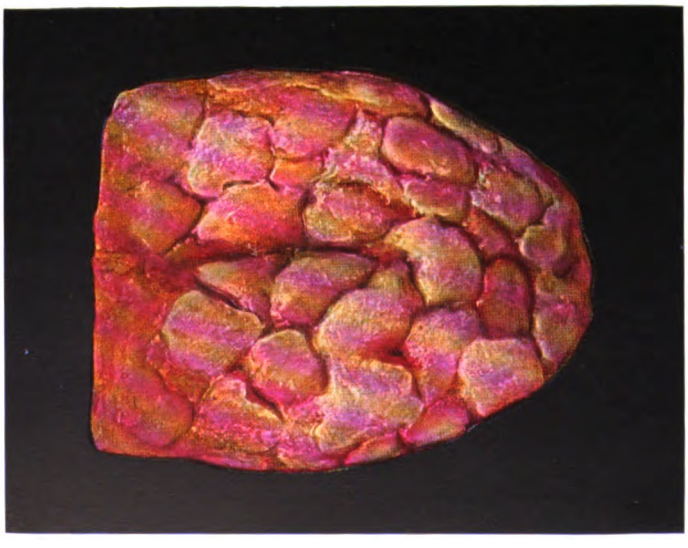

Fig. 117. Lingua geographica.

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Syphilis

SCLEROSIS SYPHILITICA LINGUÆ
(*Syphilitic Chancre of tongue*)
Plate XCIV, Fig. 118.

GUMMA LINGUÆ—LINGUA BIFIDA (*Gumma of tongue*)
Plate XCIV, Fig. 119.

GUMMA LABII SUPERIORIS ET NASI
(*Gumma of upper lip and nose*)
Plate XCV, Fig. 120.

ABSCESSUS GUMMOSI (*Gummatous Abscess*)
Plate XCV, Fig. 121.

OSTITIS GUMMOSA (*Gummatous Osteitis*)
Plate XCVI, Fig. 122.

ULCUS GUMMOSUM (*Gummatous Ulcer*)
Plate XCVII, Fig. 123.

Syphilis is a specific infectious disease which, in the great majority of cases, is contracted by sexual intercourse between human beings. It is probably caused by the *spirochaeta pallida*, which was discovered in 1905 by *Schaudinn* and *Hoffmann*, and has since been found in all the products, and also in the blood, in both acquired and hereditary syphilis. The *spirochaeta pallida* is a delicate, thin organism with corkscrew-like spirals, only visible under high magnification. It is best stained by *Giemsa's* stain or *Levaditi's* silver nitrate method.

Syphilitic infection takes place through slight excoriations or fissures of the skin or mucous membrane. In this way extragenital infection may occur in various parts of the body (lips, eyelids, tongue, nipple, fingers, etc.). Indirect contagion may also be caused by contaminated towels, linen, drinking-glasses, cigars, tobacco-pipes, shaving brushes, etc. Congenital or hereditary syphilis is the result of

syphilis in one or both of the parents. This often causes abortions or stillbirths.

In acquired syphilis, after an incubation period of three to five weeks, a circumscribed, hard, painless infiltration of the skin or mucous membrane develops at the point of infection, called the initial sclerosis or hard chancre. This forms a flat erosion with a smooth, dark-red surface, regular smooth borders and an indurated base. The chancre forms a hard nodule movable over the subjacent tissues. In genital infection it occurs on the prepuce, glans penis and labia, more rarely in the urethra; in extragenital infection it occurs at the part of the body inoculated.

In about ten per cent. of cases the chancre is not discovered, but in the genital organs of women it is often overlooked. The chancre generally heals in a few weeks (with or without treatment) and leaves a white scar which usually disappears in course of time. Suppuration only takes place when the chancre is infected by pus cocci. Sometimes the chancre becomes gangrenous (phagedenic chancre). Mixed chancre is due to simultaneous infection with syphilis and soft chancre; in these cases the soft chancre appears first and becomes indurated later on. The induration of hard chancre is due to round-celled infiltration chiefly arising in and around the walls of the small blood-vessels.

The diagnosis of chancre is usually easy when it is situated in the genital organs, but extragenital chancres are often overlooked. Chancre of the fingers often resembles a chronic whitlow or paronychia (Figs. 93 and 98); but the sore has hard borders and a smooth surface and the acute inflammatory symptoms of whitlow are absent. About a week after the appearance of the chancre the regional lymphatic glands become enlarged, forming hard, painless, movable swellings (indolent bubo). In chancre of the genitals the inguinal glands are affected; in extragenital chancre the regional glands corresponding to

the part infected. Suppuration may occur in the glands if the chancre is infected with pus cocci.

Secondary symptoms appear after a second incubation period of six to twelve weeks. They often begin with malaise, headache and pains in the joints, accompanied by a rise of temperature. A rose-red macular rash (syphilitic roseola) develops on the abdomen and thorax. Later on various syphilitic eruptions develop (secondary syphilides), the most common of which is an eruption of flat, rounded, reddish-brown or ham-colored papules situated on the trunk, face and limbs. On the forehead these papules form the so-called "corona veneris." On the genital organs and around the anus these papules become sodden and white, and are known as *condylomata lata*, which are liable to ulcerate. In some cases pustular eruptions form, and in severe or neglected cases the pustules become ulcers covered with limpet-shaped crusts (syphilitic rupia). Acneiform eruptions are common on the scalp, and scaly or psoriasiform syphilides on the palms and soles. Most secondary eruptions disappear without leaving any trace, but the ulcerative forms (rupia) leave pigmented scars, which later on become white in the center. Syphilitic eruptions are characterized by their reddish-brown or ham color, their polymorphous tendency and the absence of itching.

The mucous membranes, especially of the mouth, are affected by papular, erosive or ulcerative syphilides which are known as *mucous patches*. These develop on the tonsils, fauces, tongue, and inside the lips and cheeks, in the form of grayish-white patches or streaks, with a red border. Later on they may become eroded or ulcerated in their central parts, and then appear as red erosions with a gray border. In early secondary syphilis the tonsils and fauces may be acutely swollen (syphilitic angina), but more often there is dark-red coloration of the tonsils, fauces and soft palate. In secondary syphilis there is often loss

of hair, sometimes due to acneiform syphilides of the scalp, but more often appearing without any apparent lesion. The nails are sometimes affected with onychia or paronychia.

Secondary syphilis may last several years, and is liable to recurrences. The most contagious lesions are condylomata and mucous patches, even more contagious than the chancre.

Tertiary syphilis occurs in about twenty per cent. of cases, usually before the fifth year, sometimes later, even up to the thirtieth year after infection. The chief causes of tertiary syphilis, apart from specially virulent forms of the virus, are absence of or insufficient treatment, and abuse of alcohol.

The characteristic feature of tertiary syphilis is the formation of circumscribed or diffuse infiltrations called *gummata*. The gumma is formed of round cells, epithelioid cells and giant cells, and contains blood-vessels thickened by syphilitic arteritis. Owing to the changes in these vessels, the nutrition of the gumma is interfered with and the central parts undergo fatty degeneration or caseation. A mature gumma shows on section three zones—a central zone of caseation, a middle zone of round cells and an outer zone of fibrous tissue. Gummata may cicatrize by the formation of fibrous tissue, or they may suppurate and form an abscess. If the abscess is superficial, it breaks through the skin and gives rise to a gummatous ulcer.

It must be borne in mind that the secretion from gummatous ulcers may be contagious. (The *spirochaeta pallida* has been found in gummata, and any lesion containing this organism is contagious).

Gummata develop in the skin and subcutaneous tissue in the form of circumscribed nodules. The skin becomes reddened and may suggest a furuncle, especially in the case of a single gumma. When the gumma breaks through the skin the resulting gummatous ulcer is characteristic. The borders are hard,

smooth, not undermined but circular and sharply cut, as if punched out; the surface is covered by a tough, tenacious, yellowish deposit, or core. In the skin several gummata usually occur close together; these break down in some places and heal in others, thus giving rise to an irregular or serpiginous appearance which is characteristic of tertiary syphilitic ulceration. Gummata sometimes occur on the penis and may somewhat resemble chancres, but there is no enlargement of the lymphatic glands in gummata. Gummatous ulcers generally emit a disagreeable odor, especially when they are situated in the pharynx or nose (ozaena).

Gummata of the skin may be secondary to extension from gummata in the muscles or bones. On the other hand gumma of the skin may extend to the deeper tissues. Diffuse gummatous infiltration of the skin and subcutaneous tissue gives rise to multiple fistulas which discharge a scanty secretion. Gummata may cause extensive deformity by destruction of tissue, especially in the face (Fig. 120). Gummata of the scalp leave deep, smooth, glistening scars. Gumma of the tongue is usually situated in the center, and may divide the tongue into two parts (Fig. 119). Gummata and gummatous infiltration often affect the soft palate and pharynx, giving rise to considerable destruction of tissue and cicatricial stenosis. The larynx is also often affected. Gummatous infiltration of the rectum gives rise to stricture.

Gumma of the bones may develop in the periosteum, cortex or medulla, in the form of circumscribed growths or diffuse infiltration. Generally, all three parts of the bone are affected with simultaneous bone destruction and bone proliferation, causing an irregular, corroded appearance. Gumma of bone may undergo fibrous transformation, or may suppurate and cause necrosis. Necrosis of the cranial bones often leaves circular cavities to which the smooth, glistening skin is firmly adherent.

The nose and hard palate are often extensively destroyed by gummatous infiltration, suppuration and necrosis. The sternum and clavicle are sometimes affected. In extensive disease of the long bones curvature may result, especially outward curvature of the tibia from the weight of the body. There is also brittleness of the bones. Examination by X-rays shows irregular shadows in the periosteal region, while the cortex and medulla cannot be distinguished from one another. The whole bone is thickened and irregular.

Patients often complain of pain in the bones (osteocopic pains) before any changes are visible. Palpitation of the anterior surface of the tibia often reveals an irregular, uneven surface. The ulna, radius and fibula may also be the seat of syphilitic osteitis.

Serous effusion may occur in the joints and bursæ in the course of syphilis. Extensive disease of the joints may also arise from gummatous infiltration of the perisynovial tissue, or from gummatous osteitis of the articular ends of the bones. If a gumma of the bone breaks into the joint, suppurative arthritis generally follows. The knee joints are most often affected by syphilitic arthritis.

Gummata may occur in the muscles, and may be mistaken for tumors. They usually occur in the tongue, calf muscles and sterno-mastoid. Gumma in the brain gives rise to symptoms of cerebral tumor. Gummata are common in the liver and testicles, and may occur in the lungs, heart and other organs.

The blood-vessels are affected in all three stages of syphilis (syphilitic arteritis). The changes affect both the inner and outer coats of the vessels (endarteritis and periarteritis). Extensive proliferation of the intima may cause complete occlusion of the vessel; this occurs especially in the vessels of the brain and leads to foci of softening. Syphilitic arteritis of the aorta and other large arteries causes

aneurism. Syphilitic arteritis of the cerebral arteries causes cerebral hemorrhage.

Each of the three stages of syphilis may be absent. The chancre is undiscovered in ten per cent. of cases, and may sometimes be absent. Tertiary syphilis is said to occur in only twenty per cent. of cases; at any rate it is frequently absent. The secondary stage may also be absent in cases of severe infection in which tertiary lesions appear soon after infection (malignant syphilis). It is also possible that some cases of syphilis undergo spontaneous abortion after the chancre.

In some cases of congenital syphilis the symptoms do not appear till the eighth to sixteenth year. This is known as late or delayed hereditary syphilis, to distinguish it from early hereditary syphilis which appears at or soon after birth.

Among the characteristic signs of early hereditary syphilis are bullous syphilides of the palms and soles (syphilitic pemphigus), and epiphysitis. The latter consists in a form of osteochondritis affecting the epiphyses of the long bones, and causing thickening. It is more common in the arm and gives rise to paralysis of the limb. Epiphysitis may cause interference with growth of the limb.

In late hereditary syphilis the bones are frequently affected with gummatous processes identical with those of acquired syphilis. The tibias are often curved forwards and outwards owing to osteoplastic periostitis. This condition is known as "saber blade tibia," and is a characteristic sign of late hereditary syphilis. The skin over the bones is often ulcerated.

Syphilitic dactylitis may occur in both early and late hereditary syphilis. It causes thickening of the phalanges, usually the basal ones. It is generally multiple, sometimes bilateral, and tends to spontaneous resolution without suppuration.

The bones in hereditary syphilis are often very

brittle. Other signs of hereditary syphilis are interstitial keratitis, deafness due to disease of the internal ear, notching of the incisor teeth (*Hutchinson's* teeth). These three signs have been called the "Triad of *Hutchinson.*" Radiating scars round the mouth left by former ulcerations are also characteristic.

Acquired syphilis may also occur in infants, but differs in the absence of the characteristic features mentioned above.

Differential Diagnosis. Syphilis is so widespread among all classes of society that it must always be borne in mind in cases of doubtful diagnosis. Although the disease is fairly characteristic in all three stages, it is possible to mistake it for other affections, especially as the history can never be relied upon.

Hard chancre, when ulcerated, may be mistaken for soft chancre, but diagnosis can be established by finding the *spirochaeta pallida* in scrapings. Extragenital chancres may be mistaken for epithelioma, especially in the tongue and nipple, but the smooth surface of the chancre differs from the irregular ulcerated surface of epithelioma (cf. Fig. 1); the regional lymphatic glands are affected early in chancre. Chancre of the fingers is often mistaken for whitlow, but differs in its chronic character and absence of acute inflammatory symptoms. Secondary syphilis of the skin and mucous membranes may be mistaken for various affections, and the diagnosis often depends on the situation and general course of the lesions, and on the presence of other signs of syphilis. Gummatous ulcerations of the skin may be mistaken for tuberculous ulcers or for furuncle, but differ in the characters mentioned above. Diffuse gummatous infiltration of the skin with fungoid proliferation may suggest sarcoma (cf. Figs. 24 and 26), but differs in the absence of any tendency to bleeding, in the presence of circular scars

and brown pigmentation in the surrounding skin, and in the presence of other signs of syphilis, especially changes in the bones. Gumma in a muscle is often at first indistinguishable from a tumor. Gumma in the testicle may be mistaken for tuberculosis, but the former begins in the testicle while tubercle begins in the epididymis. The diagnosis is easy when the skin of the scrotum is perforated.

In the brain, liver, spleen and other organs the diagnosis of gumma depends on other signs of syphilis. Central gumma of bone may resemble central sarcoma or bone cyst, and may give the same appearance on X-ray examination, but gummatous changes in bone are characterized by implication of the periosteum. In doubtful cases antisyphilitic treatment should be tried. If the diagnosis hesitates between gumma and malignant tumor antisyphilitic treatment should not be continued too long, as a malignant tumor may thus become inoperable. In such cases an exploratory incision with microscopical examination is to be preferred. It must, however, be borne in mind that long-standing gumma of the skin may develop into carcinoma.

The earlier the diagnosis and the sooner the commencement of treatment, the quicker is the cure of syphilis. On the whole it may be assumed that the majority of cases become cured, but the marriage of syphilitics should not be allowed before five years after infection, and then only after thorough and prolonged treatment, with an additional course of treatment shortly before marriage. The danger of transmission to the children is diminished by time and treatment.

The disease generally runs a chronic course, and cases of acute malignant syphilis are rare except in persons who are broken down in health from other causes (tuberculosis, alcohol, etc.). In the tropics, however, syphilis is more severe and often fatal. It is also more severe in races who are attacked for the

first time and whose ancestors have been free from the disease.

In a certain number of cases syphilis causes death by gummatous disease of the internal organs, or by diseases of the nervous system, such as tabes and general paralysis, which, according to the latest researches, are always of syphilitic origin.

[Reinfection in syphilis is rare, but may sometimes occur after both the acquired and hereditary disease. Immunity in hereditary syphilis does not appear to last much beyond the age of puberty, after which acquired syphilis may be contracted, usually in an attenuated form. No doubt a soft chancre in a syphilitic subject may become indurated by the syphilitic process and be mistaken for reinfection; so may a chancriform gumma of the penis; but a considerable number of cases have been recorded in which patients passed through two distinct attacks of secondary syphilis, separated by an interval of several years. These cases must of course be distinguished from cases of relapsing secondary syphilis due to the primary infection.

Treatment. Infection can often be avoided by cleanliness—by using ointment before coitus and soap and water afterwards. Any abrasion of the epithelium of the penis, caused by balanitis, etc., may lead to infection. Antisyphilitic treatment should be commenced as soon as primary syphilis is diagnosed; it should only be delayed till secondary symptoms appear in cases of doubtful chancre. Excision of the chancre has been often tried, but it cannot prevent constitutional infection which is already present; moreover, an ulceration may occur at the place of excision. The chancre must be kept clean and dressed with iodoform, xeroform or mercurial ointment. Phagedenic chancre should be treated by prolonged immersion in mild antiseptic baths.

Treatment by mercurial inunction is one of the

best methods, and can be carried out by the patient himself. From three to five grammes (about a drachm) of *unguentum cinereum* is rubbed into the skin for about twenty minutes daily, varying the seat of inunction from day to day (inner side of arms and thighs and sides of body). This is best done at night, the patient sleeping in a flannel nightshirt and taking a hot bath in the morning. On the seventh day the patient omits the inunction. The whole course lasts six weeks. In the first year two energetic courses of inunction should be taken; in the second year two milder courses; and in the third year one course.

Treatment by intramuscular injections may be employed instead of inunction. For instance, injections of one cubic centimeter of a two to five per cent. solution of perchloride of mercury with sodiun chloride every two or three days. [Injections of perchloride of mercury are painful and have been replaced by other preparations of mercury, those most generally used being the biniodide and gray oil. Biniodide is a soluble injection given in daily injections of one-third grain. Gray oil is a preparation of metallic mercury suspended in liquid paraffin and lanolin, and is given in weekly injections of one to one and one-half grains. Injections are usually made in the gluteal muscles, but some inject into the subcutaneous tissue of the back. The treatment of average cases of syphilis can also be carried out perfectly well by internal medication in the form of pills—blue pill, proto-iodide, etc.]

Erosions and ulcerated mucous patches in the mouth may be painted with chromic acid (five to ten per cent.). To avoid mercurial stomatitis the teeth should be cleansed with carbolic tooth powder, and chlorate of potash mouth washes used.

In the tertiary stage iodide of potassium is indicated for the treatment of gummatous formations (thirty to sixty grains daily). It may be given in milk. If iodism occurs the drug should be discon-

tinued, and fifteen to thirty grains of antipyrin given daily (*Jadassohn*). If iodide cannot be borne, *Zittmann's* decoction may be tried. Hot baths and vapor baths are useful in improving metabolism, and favor the elimination of large doses of mercury.

Gummatous ulcers may be treated with iodoform, calomel ointment, or gray ointment. Gummatous abscesses may be incised and scraped. Deformities of the lips, nose, etc., caused by gummata require plastic operations. Extensive stricture of the rectum may necessitate resection of the gut. Cases of cerebral gumma, which do not yield to energetic treatment with mercurial inunction or injection and large doses of iodide of potassium, may be treated by trephining, when they cause symptoms of a circumscribed cerebral tumor. Extensive gummatous disease of the testicle may require castration. Gummata in muscles may be incised, scraped and treated locally with mercurial ointment, if they do not yield to general antisyphilitic treatment. The same applies to gummata in the bones, especially when they cause severe pain. Gummatous periostitis and osteitis often heal under energetic antisyphilitic treatment, but sometimes require operative treatment for the removal of sequestra. In cases of delayed union of fractures, iodide of potassium is often useful when there is a history of previous syphilis. The same applies to all badly healing wounds in syphilitic patients, especially operation wounds. In hereditary syphilis, osteochondritis can be treated by splints, and gummatous osteitis may eventually require operative interference.*

* For further information on this subject the reader is referred to *Marshall's* "Syphilology and Venereal Disease," London. Balliére, Tindall and Cox; *Marshall's* "Golden Rules of Venereal Disease," Bristol. John Wright and Co.; *Marshall's* translation of *Fournier's* "Treatment and Prophylaxis of Syphilis," New York. Rebman Co.

SCLEROSIS SYPHILITICA LINGUÆ

(*Syphilitic chancre of the tongue*)

Plate XCIV, Fig. 118.

This is a case of extragenital chancre affecting the tongue. The sore is slightly raised above the surface; it has a round form with hard, slightly raised not undermined borders, and a smooth, varnished surface. The lymphatic glands in the submaxillary and occipital regions were hard and movable. Carcinoma of the tongue differs from this in its irregular surface, from which epithelial plugs can be expressed, and in the glandular affection occurring later.

As already mentioned, syphilitic contagion may take place through intermediate objects. *Von Bergmann* has observed a case in which contagion was due to smoking the fag end of a cigarette thrown away by a syphilitic person.

GUMMA LINGUÆ—LINGUA BIFIDA (*Gumma of the tongue*)
Plate XCIV, Fig. 119.

Gumma of the tongue is usually situated in the center of the tongue, while carcinoma generally affects the posterior part of the side of the tongue (Fig. 9). A breaking-down gumma may divide the tongue into two parts (bifid tongue). The figure shows a broken-down gumma with its characteristic tenacious, yellowish-brown deposit. Syphilitic infection was denied in this case, but it was cured by antisyphilitic treatment.

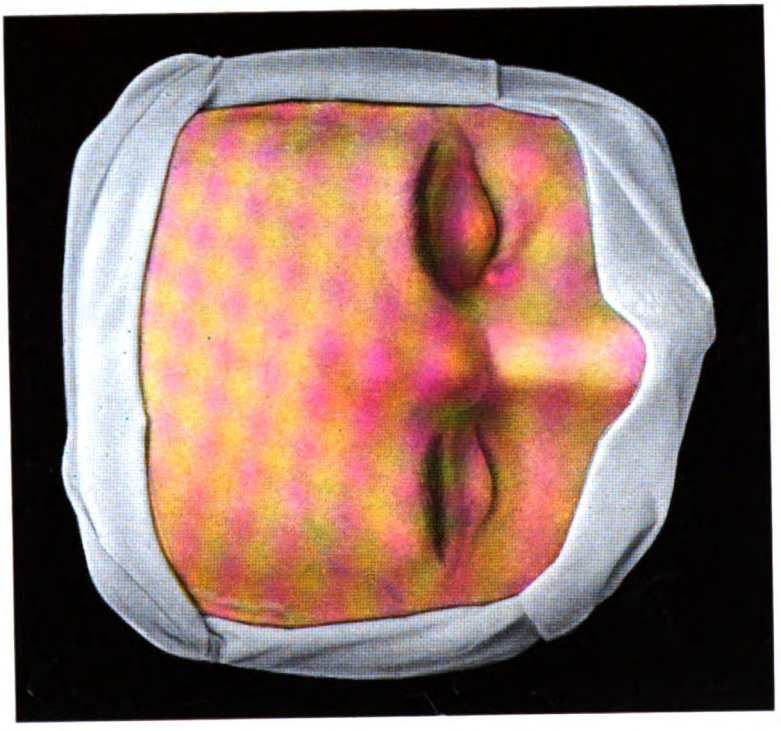

Fig. 121. Abscessus gummosi.

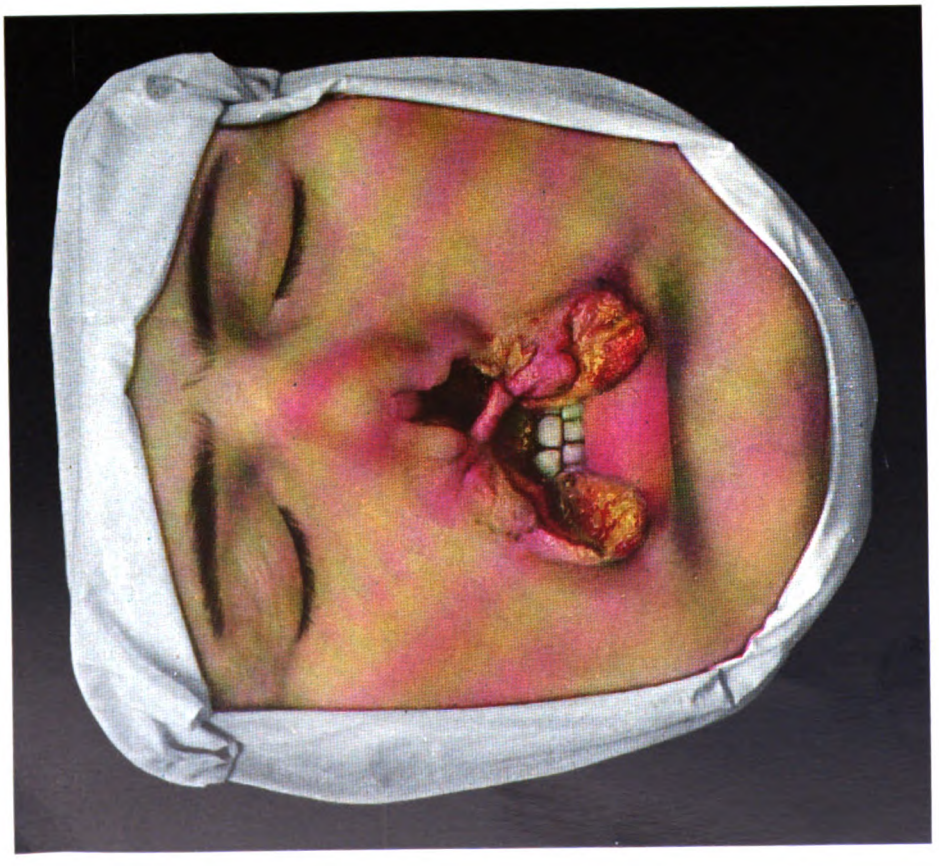

Fig. 120. Gumma labii superioris et nasi.

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GUMMA LABII SUPERIORIS ET NASI

(*Gumma of the upper lip and nose*)

Plate XCV, Fig. 120.

This case shows extensive destruction of the upper lip, the cartilaginous portion of the nose, the nasal septum, and the bony framework of the nose, due to gummatous ulceration. There is also perforation of the hard palate. The upper lip shows the characteristic yellow, tenacious deposit, which can be removed without bleeding. The surface of the ulceration is fairly smooth and the borders soft, as compared with the irregular surface and hard borders of carcinomatous ulceration. The patient had syphilis ten years previously.

The patient was treated with iodide internally and mercurial ointment locally. After the ulcerated surface had become clean, the borders were excised and united by sutures. The defect in the nose was repaired by a plastic operation, and an obturator was worn for the perforation in the palate.

ABSCESSUS GUMMOSI (*Gummatous Abscess*)
Plate XCV, Fig. 121.

This is a case of multiple gummata in the skin of the face, situated at the root of the nose, in the left eyelid and in the temporal region. The skin is thin and red. Fluctuation was felt on palpation. The patient could not remember contracting syphilis, but his wife had had frequent abortions and several syphilitic children. There were gummatous processes in the skin of various parts of the body; also limitation of movement in the elbow joint due to previous gumma of the bone. The surface of both tibias was irregular, and there were circular scars on the legs. The patient also suffered from severe headache and attacks of giddiness, due to syphilitic disease of the cerebral arteries.

The abscesses were incised and scraped, and healed under antisyphilitic treatment. The cerebral symptoms also improved.

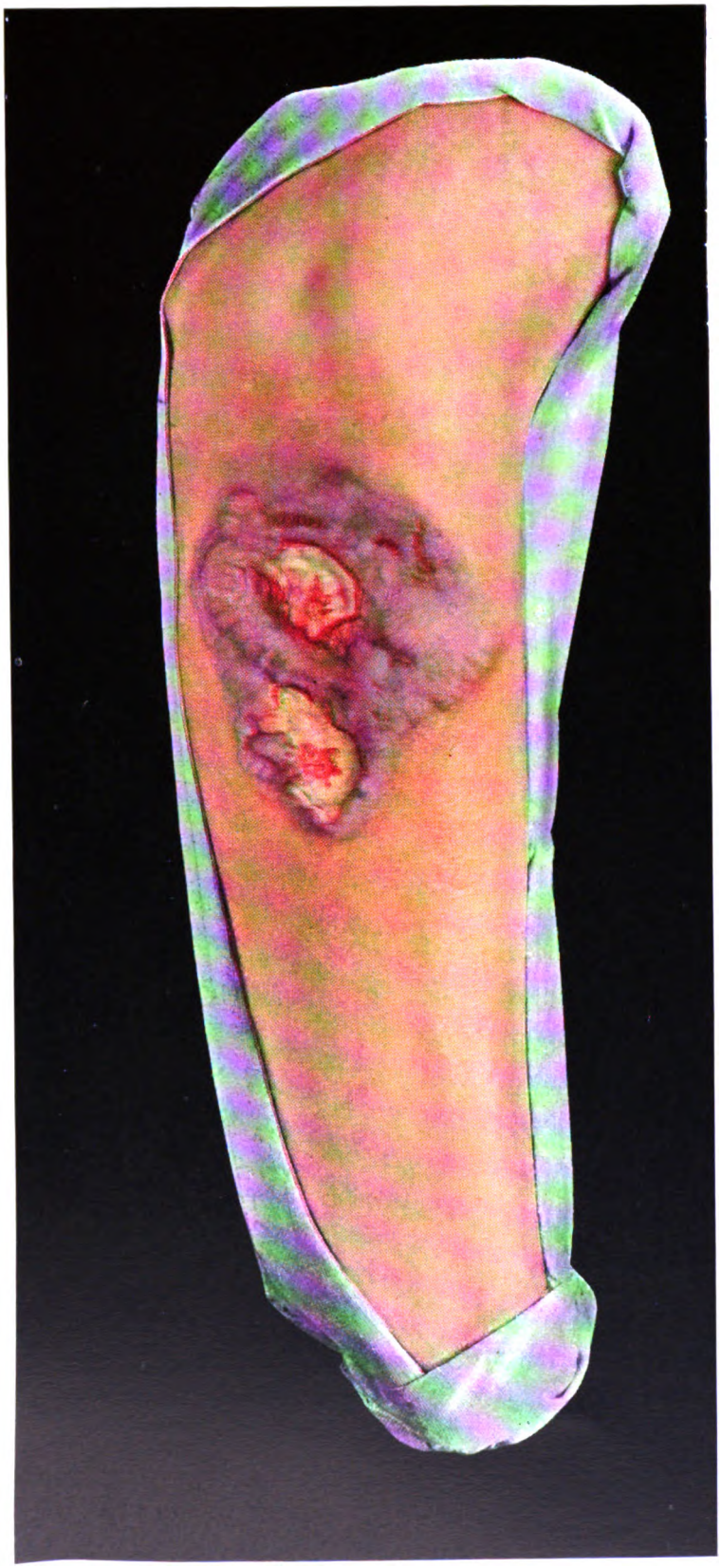

Fig. 122. Ostitis gummosa.

Rebman Company, New-York.

OSTITIS GUMMOSA (*Gummatous Osteitis*)
Plate XCVI, Fig. 122.

This patient acquired syphilis twenty years ago, and suffered for some years from pain in the right forearm, especially at night. The bones of the forearm gradually became thickened, and the skin red and swollen. Two irregular ulcers developed, covered with yellow, tenacious deposit. Round the ulcers soft proliferations formed resembling sarcomatous tissue. There were several small fistulas leading to the bones, in which the X-rays showed irregular proliferation of the periosteum and irregular thickening of the cortex. Healing gradually took place under treatment by iodide internally and mercurial ointment locally. There were no other signs of syphilis.

ULCUS GUMMOSUM (*Gummatous Ulcer*)
Plate XCVII, Fig. 123.

In this case a gumma occurred in the skin over the internal malleolus after a kick (trauma is sometimes an exciting cause of gumma). The skin became infiltrated, swollen and red, and gradually broke down, forming an ulcer with sharply cut edges and a base covered with tenacious, yellow deposit. The patient contracted a sore on the penis some years previously, which was diagnosed as a soft chancre, and received no specific treatment. Three years after infection a gumma developed in this situation and was treated for a long time with poultices, but was afterwards healed by iodide of potassium internally and mercurial ointment locally. The patient was recommended further treatment by mercurial inunction or injections.

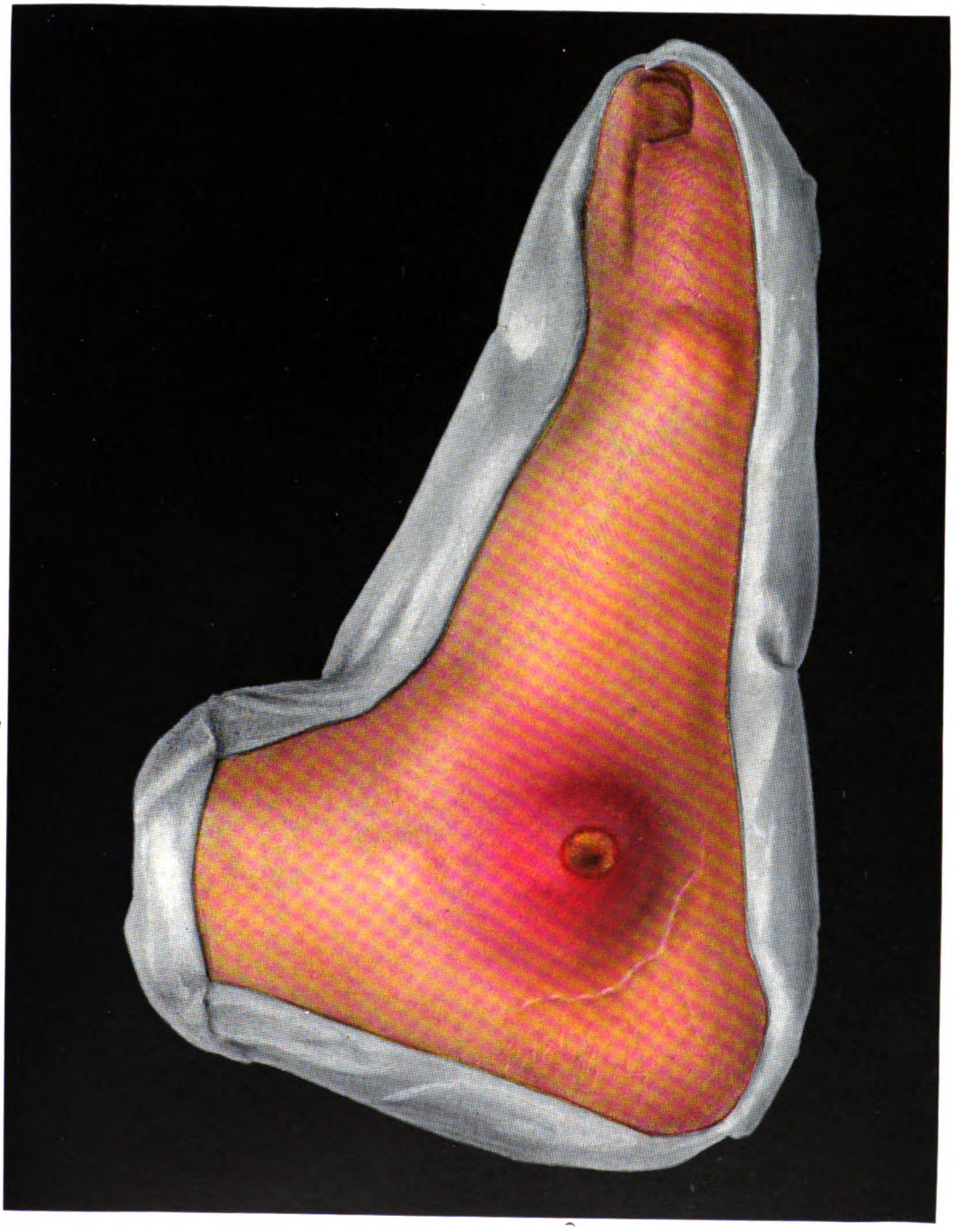

Fig. 123. Ulcus gummosum.

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Tuberculosis

LYMPHOMATA COLLI TUBERCULOSA
(*Tuberculous lymphoma of the Neck*)
Plate XCVIII, Fig. 124.

ARTHRITIS TUBERCULOSA FUNGOSA
(*Fungating Tuberculous Arthritis*)

ANKYLOSIS GENUS FIBROSA
ABSCESSUS FRIGIDUS
(*Fibrous Anchylosis of the Knee—Cold Abscess*)
Plate XCIX, Fig. 125.

ARTHRITIS TUBERCULOSA PURULENTA
(*Purulent Tuberculous Arthritis*)
Plate C, Fig. 126.

ARTHRITIS TUBERCULOSA FIBROSA
(*Fibrous Tuberculous Arthritis*)

ANKYLOSIS OSSEA—SUBLAXATIO
(*Bony Anchylosis—Subluxation*)
Plate C, Fig. 127.

ARTHRITIS TUBERCULOSA FIBROSA
(*Fibrous Tuberculous Arthritis*)

TUMOR ALBUS (*White swelling*)
Plate CI, Fig. 128.

TUBERCULOSIS TESTIS (*Tuberculous Testicle*)
Plate CII, Fig. 129.

TUBERCULOSIS MANUS (*Tuberculosis of the Hand*)
Plate CIII, Fig. 130.

OSTITIS TUBERCULOSA (*Tuberculous Osteitis*)
SPINA VENTOSA (*Spina ventosa*)
Plate CIV, Fig. 131.

GANGRÆNA PEDIS HUMIDA (*Moist Gangrene of the Foot*)
Plate CIV, Fig. 132.

Since the discovery of the tubercle bacillus by *Robert Koch*, in 1881, it is known that tuberculous affections are solely due to the invasion of these bacilli; although tuberculosis was regarded as an infective disease by several investigators before the time of *Koch*. *Baumgarten* also discovered the tubercle bacillus almost at the same time as *Koch*.

The tubercle bacilli are straight or slightly curved rods. They are easily stained by the *Ziehl-Neelsen* method, or by *Gram's* method, and are not decolorized by nitric acid solution (acid fast bacilli). The bacilli retain their virulence for a long time in the dry state, but are destroyed by boiling and by sunlight. Besides microscopic examination and culture of the bacilli, inoculation of the guinea pig is useful for establishing diagnosis. Recent researches by *Friedrich* have shown that tubercle bacilli in cultures assume the form of club-shaped radiating filaments similar to actinomyces; so that the bacillary nature of the tubercle bacillus is doubtful, and it may belong to the hyphomycetes. In any case tuberculosis and actinomycotic affections are often very similar.

Tubercle bacilli are present in dust and on the walls of rooms. Patients with tuberculosis of the lungs infect the air with small vesicles of fluid containing tubercle bacilli. Tubercle bacilli may also penetrate the unbroken skin and mucous membrane and cause infection of the lymphatic glands; but this form of infection is comparatively rare. Wounds are easily infected with tubercle bacilli, especially when the sputum of a tuberculous subject comes in contact with a wound (*e.g.* tattooing). Although, according to *Koch*, there may be a distinction between human and bovine tuberculosis, the latter may be transmitted to man, and infection may occur from the meat and milk of animals infected with bovine tuberculosis; [especially by milk from cows with tuberculous udders].

As regards the hereditary transmission of tuberculosis, it is certain that the children of tuberculous parents are more predisposed to tuberculosis than the children of healthy parents; but, whether the bacilli can be transmitted from the mother to the fetus and remain for a long time latent in the tissues of the child, and whether transmission can take place through the semen of the father, are points which are

still unsettled. In distinction to hereditary predisposition, there is acquired predisposition; after certain diseases, such as influenza, measles, bronchial catarrh and glandular swellings. The stronger the body at the time of infection, the more it is able to resist the disease. It is said that signs of former tuberculosis can be found in almost ninety per cent. of all men, in a great many of these cases the tuberculous foci having become encapsuled or calcified. Bad feeding, unhealthy dwellings, sedentary occupations and alcoholism predispose to infection.

In the great majority of cases primary tuberculosis affects the lungs, either by direct inhalation of bacilli, or by bacillary infection of the lymphatic glands (tonsils, bronchial glands, nasopharyngeal glands). If the bacilli remain in the lungs they give rise to phthisis. Tuberculosis of the mouth, pharynx, larynx, trachea, bones and all other tissues, is in most cases due to secondary metastatic infection by the blood. Tuberculous embolism may be caused by a tuberculous focus breaking through a large vessel.

Tuberculous lesions which interest the surgeon are in the majority of cases secondary. Tuberculosis may attack any of the tissues, but has a predilection for certain ones—primary tuberculosis for the lymphatic glands and lungs; secondary tuberculosis for the bones and joints. Tuberculosis of the intestine, which generally affects the small intestine and ileocæcal region, is rarely primary but generally secondary to tuberculosis of the lung (by swallowing phthisical sputum) or the mesenteric glands. Tuberculosis may occur at any age.

The tubercle bacilli give rise to small nodular infiltrations known as tubercles or granulomas. The granuloma is characterized by the presence of several forms of cells, the majority of which are round cells, a smaller number epithelioid cells, in fresh tubercles; while in older tubercles giant cells are present,

especially in tubercles with a tendency to heal. The giant cells of tubercle differ from other giant cells in the fact that the nuclei are situated at the periphery of the cell round a central homogeneous mass, and that in some parts of the circumference of the cell there is a double arrangement of nuclei. Owing to the absence of blood-vessels in the center of the tubercle there is caseous degeneration of the central cells. The giant cells often contain tubercle bacilli and are believed to take part in the process of healing, by acting as phagocytes.

The tubercle sets up inflammatory reaction in the surrounding tissues, resulting in the formation of granulation tissue and pus, the latter being discharged by a fistula, or forming an ulcer when the process is in the skin. In most cases the body tries to expel the tuberculous focus, but in some cases the latter becomes encapsuled by connective tissue. This connective-tissue capsule may at any time be ruptured, by trauma, etc., and give rise to fresh tuberculous infection. The majority of cases of tuberculosis following an injury are explained by the setting free of encapsuled foci of tubercle; this not only causes a fresh outbreak of tubercle at the seat of the injury, but also spread of the previously encapsuled focus of disease to other organs.

Surgical cases of tuberculosis are generally characterized by the formation of typical granulations, fistulas and specific pus. The granulations are pale and vitreous. The fistulas run an irregular course, and, in cases of tuberculous bone disease, open at more or less distant points in the skin; the walls of the fistula are soft and bleed easily. The pus is thin and mixed with fibrin, caseous masses and shreds of tissue. The tuberculous ulcer is characterized by thin, soft, ragged, undermined borders, and a base covered with yellow caseous masses, or pale-red or gray granulations. Tuberculous granulations may destroy all the surrounding tissues (bones, cartilage

and muscles) and the necrosed parts are expelled from the body. In the majority of cases there is a formation of soft, spongy granulations, and little fibrous tissue formation. Tuberculous processes often continue for years before an abscess forms, or a cavity from destruction of the tubercle.

Tuberculosis affects the different tissues in characteristic ways, which we shall describe later when dealing with the different cases. As a rule it runs a chronic course with intermittent fever, without acute inflammatory symptoms. The diagnosis can often be made from the appearance of the ulcer, fistula, pus or granulation tissue, and by the X-rays in the case of bone disease. In many cases tuberculosis of the lung leads to tuberculous disease of other tissues. Diagnosis can be confirmed by microscopic examination; or by inoculation of suspected tissue into the peritoneum of the guinea-pig, which gives rise to tuberculosis of the mesenteric glands in a few weeks. The walls of tuberculous abscesses are very suitable for inoculation, as they contain many tubercle bacilli.

The prognosis depends on the situation and extent of the disease. Small, circumscribed foci can be radically removed by operation—for instance, circumscribed tuberculosis of the skin, or tuberculous glands in the neck which are common in children. However, as tubercle is generally present in the lungs, many patients succumb to this sooner or later. In tuberculosis of bones and joints, complete restitution is seldom possible, owing to the extensive destruction of tissue. Long-standing disease of bones and joints, which may occur at an advanced age, is often fatal from exacerbation of tuberculosis of the lungs.

Treatment. The spread of the disease should be checked by prophylactic measures. Tuberculous patients should be warned against spitting into handkerchiefs or on the ground, and should use spitting-cups. Meat and milk from tuberculous cattle should

not be consumed. The general treatment of tuberculous patients consists in nourishing diet (plenty of milk, meat and butter), cod-liver oil and extract of malt; administration of preparations such as creosote and guiacol; residence at high altitudes; sea baths; sanatorium treatment. Inunction of the whole body with green soap, which is allowed to remain on the skin for half an hour, is said to be beneficial. Brine baths and sulphur baths are useful. Tuberculin treatment has not been successful, and cannot be recommended in practice.*

As regards local treatment, the object of modern surgery is to remove the focus of disease when it is within reach. By this means not only is the local disease often cured, but the primary lung disease is often improved. In some cases a whole organ, such as the kidney or testicle, must be removed when it is extensively diseased. A ten per cent. emulsion of iodoform in glycerin is useful for application to tuberculous ulcers and fistulas, and for injection into tuberculous joints and abscesses. In the treatment of tuberculous bone and joint disease immobilization is essential.

Tuberculosis of the Skin. This has already been mentioned in the case of lupus. (Plate III.) Local tuberculosis of the skin may occur from infection from dead bodies affected with tuberculosis. This form is common on the fingers in doctors and hospital attendants after post-mortem examinations, and in butchers from handling tuberculous meat. It is known as cadaveric tubercle or post-mortem wart. It commences as a small red spot which develops into a raised nodule with slight sanious discharge. Several nodules may develop close together and form a warty growth. A more extensive

* Sir Almroth Wright's method by injection of the new tuberculin, under control of the opsonic index, is apparently successful in suitable cases.

form of warty cutaneous tuberculosis is known as *tuberculosis verrucosa*, and is common in the poorer classes. In these cases the tubercles have little tendency to break down and undergo caseous degeneration, but become warty by cornification of the epidermis.

The treatment of these forms of cutaneous tuberculosis is the same as for lupus; *viz.* excision of small lesions; scraping with sharp spoon, cauterization, or treatment by *Finsen's* light in the case of larger growths.

Another form of cutaneous tuberculosis commences in the subcutaneous tissue, and gradually extends to the skin in the form of red nodules resembling furunculous abscesses. The skin becomes thin, the nodules suppurate and discharge pus on the surface. This condition is common in the neck in tuberculous children. Before the skin breaks down the nodules may be mistaken for gummata, but afterwards typical tuberculous ulcers are formed. This condition has been called scrofuloderma, but is due to the action of tubercle bacilli. The treatment consists in scraping and iodoform dressings.

Tuberculous Lymphangitis and Lymphadenitis. Tuberculous lymphangitis is rare, and only occurs in connection with tuberculosis of the skin and lymphatic glands, in the form of nodular cords. Tuberculous lymphadenitis, on the other hand, is very common (Fig. 124). It occurs especially in children in the glands of the neck, the tubercle bacilli easily penetrating the soft walls of the lymphatic vessels. The glands may be affected by way of the blood or lymph, after eczema, ulcers or tuberculosis of the neighboring tissues. Through slight lesions of the mucous membrane of the mouth or pharynx, the tubercle bacilli enter the lymphatics, and infect the glands of the neck and submaxillary region. Some authorities maintain that tuberculosis

of the lungs is secondary to tuberculous disease of the bronchial glands, and intestinal tuberculosis to disease of the mesenteric glands. The tubercle bacilli cause inflammatory swelling, and the formation of miliary tubercles in the glands. Several miliary tubercles become confluent and form larger nodules which undergo caseous degeneration and softening, and finally suppurate. The tuberculous process is not usually limited to a single gland, but extends through the capsule to the surrounding tissue, and finally to the skin. The glandular tumor, at first circumscribed and covered with intact skin, soon implicates the skin and breaks through it in one or more places, forming fistulas which discharge thin, greenish pus. The pus often burrows under the skin and breaks through in more or less remote places. The axillary and inguinal glands are seldom the seat of primary tuberculosis.

Differential Diagnosis. Tuberculous glands are characterized by the variation in their consistence; some glands being soft and fluctuating, others hard. In the absence of fistulas or other signs of tuberculosis, an isolated tuberculous gland may be mistaken for a suppurating sebaceous cyst or dermoid. The differential diagnosis from malignant tumors has already been described (Fig. 24). In doubtful cases microscopic examination, or inoculation in the guinea pig will establish the diagnosis.

Treatment. The primary cause (eczema, ulcers, etc.), must, of course, be treated. Circumscribed glandular abscesses may be evacuated by puncture and injected with ten per cent. iodoform emulsion. Larger groups of glands should be freely laid open and removed. Removal of tuberculous glands in the neck requires an accurate knowledge of anatomy, as these glands are often situated around the large

vessels from the mastoid process to the supraclavicular fossa, and lie behind the sterno-mastoid muscle and sometimes under the trapezius. After extirpation, the wound should be plugged with iodoform gauze and the wound closed, leaving a small space for drainage. In children especially, there is a rise of temperature for the first few days after extensive removal of glands, which is probably due to the entrance of tubercle bacilli into the blood. Miliary tuberculosis may develop after extensive removal of tuberculous glands. It is, therefore, better in extensive glandular disease, occurring in feeble patients, to limit operative interference to incision and scraping.

Tuberculous lymphadenitis of the neck, especially when associated with eczema of the eyelids, otitis media and ulcers of the cornea, is often wrongly called scrofula. Staphylococci are often found along with tubercle bacilli. In cases where no tubercle bacilli are found it is possible that they have been destroyed by the pus cocci. The term scrofula should, therefore, be avoided, especially when typical tuberculous disease is present in other parts of the body. Predisposing causes of tuberculous lymphadenitis are—measles, influenza, whooping-cough, uncleanliness and improper feeding. General treatment consists in the measures already mentioned, especially sea and sulphur baths.

Tuberculosis of Bone. Tuberculous disease of bones is secondary, and caused by the spread of tuberculous material by way of the blood. For this reason the bones are generally affected in certain places corresponding to the distribution of their blood-vessels. *Lexer* found, by X-ray examination after injection of the vessels of bones with mercury, that the nutrient artery of the long bones terminated in the epiphyses. This explains the frequency with which the epiphyses of the long bones are affected

with tuberculous deposits, by plugging the terminal branches of the nutrient artery in the epiphysis with tuberculous infarcts. In the short bones the nutrient artery terminates soon after its entrance in the middle of the diaphysis; hence tuberculous disease of these bones affects the diaphysis. Tuberculosis also affects the vertebræ, the bones of the hand and foot, the cranial bones, the sternum, ribs and ilium.

In most cases there is circumscribed disease in the form of a caseous sequestrum. Around this form granulation tissue and pus, which seeks a way to the surface by the formation of a fistula. Small sequestra often give rise to large abscesses which become visible under the skin, often at some distance; these are known as "cold abscesses" (Fig. 125). Tuberculosis of the vertebræ may thus cause abscesses which appear in the thigh. In tuberculous bone disease there is little tendency to the formation of new bone. In some cases the focus of disease may become encapsuled in the bone, but is always liable to recrudescence, especially after an injury. More commonly the sequestrum is discharged piecemeal through a fistula, thus differing from the large sequestrum of pyogenic osteomyelitis. Multiple foci of disease often occur in one or more bones. When the bone is exposed by incision, irregular, caseous fragments are seen, together with pus. When the disease occurs in the epiphyses of the long bones it may break into the joint, giving rise to suppurative arthritis.

Although the foci of disease are usually small, and there is seldom the necrosis of large portions of bone which occurs in pyogenic osteomyelitis, there may be extensive disease of the medullary cavity when tuberculous disease of a joint extends to the bones. The phalanges may also be extensively diseased. Sometimes no changes are found in the bone, and the disease is confined to the periosteum, giving rise to subperiosteal abscess, especially in the ribs.

Differential Diagnosis. In some cases tuberculosis of bone may be mistaken for the chronic forms of pyogenic osteomyelitis. However, tuberculous bone disease can nearly always be recognized by its typical situations, its characteristic pus, its small sequestra, its slight tendency to new bone formation, and by the presence of tuberculosis of the lungs. In many cases the diagnosis is assisted by the X-rays. Some cases may be mistaken for syphilitic bone disease. In doubtful cases diagnosis can be settled by incision.

Treatment. As soon as tuberculous disease of bone is diagnosed (by the X-rays early diagnosis can be made), the disease must be radically removed, without interfering too much with function. The bone must be freely exposed, the diseased parts removed by the gouge or sharp spoon, and the wound plugged for some time with iodoform gauze. In the extremities immobilization is necessary. Later on, iodoform-glycerin emulsion may be injected into the bone cavity. Tuberculous foci can be treated in this way in the bones of the face, the cranial bones, the sternum and the ilium.

In cases of tuberculous disease of the vertebræ (tuberculous spondylitis or *Pott's* disease) operative interference should be limited to the evacuation of abscesses, which often point on the inner side of the thigh below *Poupart's* ligament, and injection of iodoform-glycerin emulsion. Operation on the vertebræ themselves is likely to injure the spinal cord or nerves. These cases often undergo spontaneous cure by sinking of the bodies of the vertebræ, resulting in kyphosis. These cases require immobilization by extension splints and later on by plaster of Paris jackets. Extensive bone disease of the extremities in old people may require amputation to save the patient from death by pulmonary tuberculosis; on which amputation often has a favorable influence.

Treatment of tuberculous bone disease by passive hyperæmia is only of use when combined with other methods of treatment. In cases of pain and fatigue in the limbs of young people, occurring without apparent cause, the possibility of commencing tuberculous disease of the bones or joints must always be borne in mind. Early cases often recover after prolonged immobilization without operation. Bones which have been affected by tuberculosis must be protected from injury, which may start the disease afresh.

Tuberculosis of Joints. The joints are often affected with tuberculosis, generally by extension from tuberculosis of the bones. Infection of the joints may also take place through the blood, but primary tuberculosis of joints is rare. In most cases both the synovial membrane and the articular ends of the bones are affected. The knee and hip joints are most often attacked; after these the wrist and elbow. Tuberculous joint disease is most common before puberty, but it also occurs at an advanced age.

The tubercle bacilli give rise to the formation of granulation tissue and effusion in the joint. In the mildest forms there may be only serous effusion (hydrops), but more commonly the effusion is serofibrinous. The fibrin forms villous deposits on the synovial membrane and cartilage, and the so-called "rice bodies," which are lumps of loose fibrin in the joint. These milder forms of joint disease may be included under the name of *articular hydrops*.

The second form of tuberculous arthritis is known as *fungoid arthritis*, owing to the formation of fungoid or spongy granulation tissue, which gives rise to globular swelling of the joint. In these cases the whole joint is filled with grayish-red or yellowish-white granulations, and there is only slight exudation. The fungous granulations tend towards caseous degeneration, and after a time to suppuration. This

form of tuberculous arthritis does not remain limited to the joint but soon extends to the ligaments and periarticular tissue, and eventually to the subcutaneous tissue and skin (Figs. 125 and 126).

A third form is *fibrous arthritis*, in which there is a formation of hard fibrous tissue in the joint. This form is called *caries sicca* by *Volkmann*. It is common in the shoulder and hip joints, and is characterized by a great tendency to cause atrophy, of the articular end of the bone, giving rise to dislocations and also to muscular atrophy.

In distinction to the above atrophic form, there is another form of fibrous arthritis causing globular swelling of the joint from the abundant formation of fibrous tissue. This is especially common in the knee joint and may be mistaken for bone tumor. It is known as "white swelling" or *tumor albus*, owing to the white anæmic appearance caused by pressure of the fibrous tissue on the skin.

A fourth form of tuberculous joint disease is *purulent arthritis*. This is often due to mixed infection of one of the above-mentioned forms with staphylococci—for example, through a fistula in the skin. However, purulent arthritis sometimes occurs quite suddenly, especially in children.

In all these forms of tuberculous arthritis the cartilage may be destroyed by the fibrinous exudation. In cases of fibrinous hydrops, and in caries sicca, the destructive action is generally limited to the cartilage; but in the fungoid and purulent forms of arthritis the whole epiphysis may be destroyed, and the infection may spread to the diaphysis. Besides this, multiple abscesses often develop at some distance from the joint. The greater the destruction of the joint the more abnormal are the positions of the affected limb. The affected joint assumes the position in which its capsule has the greatest capacity (*i.e.* the position in which the capsule is fully distended). For this reason the knee joint is in the position of flexion, the hip

joint in the position of abduction and flexion, the elbow joint in the position of flexion, and the shoulder joint in the position of external rotation. Fibrous or bony anchylosis may occur in these positions; also in positions of subluxation or dislocation.

Tuberculous arthritis generally begins with pain, which is often remote from the affected joint; *e.g.* in disease of the hip joint pain is referred to the inner side of the knee. This is followed by slight rises of temperature and pain in the region of the affected joint. Movement of the joint is avoided, the whole joint becomes swollen, and characteristic positions are assumed by the different joints. In hydrops there is fluctuation. In fungoid arthritis the whole joint is filled with soft, spongy tissue, causing balloon-like swelling of the joint (ballooning); this spongy tissue extends to the periarticular tissue and reaches the skin, which becomes reddish blue, and later on breaks down into tuberculous ulcers and fistulas (Fig. 125).

Diagnosis is generally easy in cases with a fistula discharging characteristic thin tuberculous pus mixed with caseous débris and fragments of sequestrum. In other cases there is evidence of tuberculosis in the lungs or other organs. The fibrous forms (caries sicca) are characterized by the marked atrophy of the joint, the abnormal positions, and the muscular atrophy and complete loss of function. White swelling is recognized by the extensive tumor-like swelling covered by white skin (Fig. 128). In purulent arthritis there is redness and swelling of the skin with high temperature. In doubtful cases an incision will make the diagnosis clear.

Differential Diagnosis. Tuberculous hydrops may be mistaken for traumatic effusion, gonorrheal arthritis or syphilitic arthritis. The diagnosis depends on the history of the case and thorough examination of the whole body. In doubtful cases the joint may be punctured, or inoculation of the

guinea pig may be performed. Acute forms of fungoid tuberculous arthritis can hardly be mistaken for other affections. In cases where complete healing of the joint has taken place, with bony anchylosis, it is sometimes impossible to distinguish tuberculous cases from joint disease secondary to pyogenic osteomyelitis of the diaphysis. In old people healed tuberculous joints may be mistaken for arthritis deformans or chronic rheumatism. Purulent tuberculous arthritis often resembles acute pyogenic osteomyelitis. In young children especially, when the disease begins with rigors, high fever and constitutional disturbance, diagnosis is often only made after incision.

The prognosis of tuberculous arthritis is more favorable in young individuals than in old people. Chronic tuberculous arthritis may give rise to miliary tuberculosis, or to amyloid degeneration of the internal organs.

Treatment. In its early stages tuberculous arthritis may be cured by immobilization by means of extension splints or plaster of Paris casings. Conservative treatment should always be adopted in the early stages. Hydrops may be treated by repeated puncture, injection of iodoform-glycerin emulsion or alcohol and immobilization of the joint. Recurrence is common, and complete restoration of function seldom occurs. The joints should, therefore, be allowed to anchylose in the most useful position. When abscesses and fistulas form, and when an extensive focus of bone disease is shown by the X-rays, conservative treatment must be abandoned.

In fibrous arthritis, caries sicca and white swelling, resection of the joint should be performed as early as possible, to prevent muscular atrophy. In the shoulder joint resection gives good results; but in the knee joint, bony anchylosis in the straight position is the only possible result. In fungous arthritis,

especially in young patients, operation may be limited to opening the joint and carefully removing all tuberculous disease (arthrectomy). The capsule of the joint must be excised wherever it is diseased, and tuberculous foci in the cartilage and bone removed with the gouge. In young subjects a typical resection of the joint is to be avoided, owing to interference with the growth of the limb by extensive removal of the epiphyses.

In adults, on the other hand, the joint may be resected and all diseased parts carefully removed. If the medullary cavity is found to be diseased, after resection of the epiphysis, it must be scraped out. Abscesses and fistulas require incision and scraping. In purulent arthritis the joint must be freely opened; in advanced cases resection is necessary. In extensive tuberculous arthritis with tuberculous disease of the neighboring bones and soft parts, amputation may be necessary, especially in old people (Fig. 130).

After operation the joint must be plugged with iodoform gauze, drained, and immobilized. Joints which have become healed in abnormal positions may be forcibly corrected under an anæsthetic when the anchylosis is fibrous; but there is danger of rupture of the vessels and consequent gangrene (Fig. 132). It is better to treat fibrous anchylosis by gradual extension; while bony anchylosis in a bad position may require resection.

After operations on joints, these should be protected by light splints (*e.g.* poroplastic casings) till the end of the period of growth in children, and for some years in adults. The disadvantage of this apparatus is the causation of muscular atrophy. On the other hand, after resection of the knee joint in young subjects, the knee often becomes flexed, even after bony anchylosis, requiring further resection.

Tuberculosis of other Tissues. Tuberculosis of the mucous membranes occurs in the buccal cavity, the tongue, lips, larynx, small intestine and rectum,

and is generally secondary to tuberculosis of the lungs. *Von Bergmann* has observed a case of tubercular infection of the mouth, from a culture of tubercle bacilli, which was cured by excision. Tuberculosis of the mucous membranes develops in the form of small, reddish-gray nodules, which break down into small easily bleeding ulcers with ragged edges and a yellow caseous surface. These are best treated by cauterization with strong lactic acid. Fistula of the rectum, which is common in intestinal tuberculosis, requires incision. Tuberculosis of the ileo-cæcal region, causing fibrous stricture, may require resection of the gut.

Tuberculous peritonitis, which gives rise to exudation and the formation of extensive adhesions, is improved by laparotomy and removal of the exudation. Purulent tuberculous effusion into the pleural cavity should be evacuated by resection of the ribs. Tuberculosis of the testicles and kidneys necessitates removal of these organs. Tuberculosis of the bladder should be treated by irrigation and the internal administration of guiacol. It has been attempted to remove isolated foci of tuberculosis in the lungs by operation.

Treatment of the general condition of the patient is necessary in all forms of tuberculous disease.

Miliary tuberculosis, which may develop after extensive operations, such as removal of tuberculous glands in the neck, or after breaking down joint adhesions, is due to dissemination of tubercle bacilli in the blood, and may take the form of a typhoid condition, pulmonary disease or meningitis. It is not amenable to surgical treatment.

LYMPHOMATA COLLI TUBERCULOSA

(*Tuberculous Lymphoma of the Neck*)

Plate XCVIII, Fig. 124.

This is a case of tuberculosis of the submaxillary and cervical glands. The patient suffered since youth from eczema of the face and inflammation of the eyelids. A swelling gradually formed in the neck over which the skin became livid. A series of swellings of different sizes were felt under the skin, which was movable over them. Some of these were hard, others soft and fluctuating. There was no sign of pulmonary tuberculosis. The glands were removed through an incision along the inner border of the sterno-mastoid. In removing glands with suppuration in their interior, care must be taken not to break into them and thus infect the wound. The wound was plugged with iodoform gauze and sutured, leaving a space for drainage at the lower end.

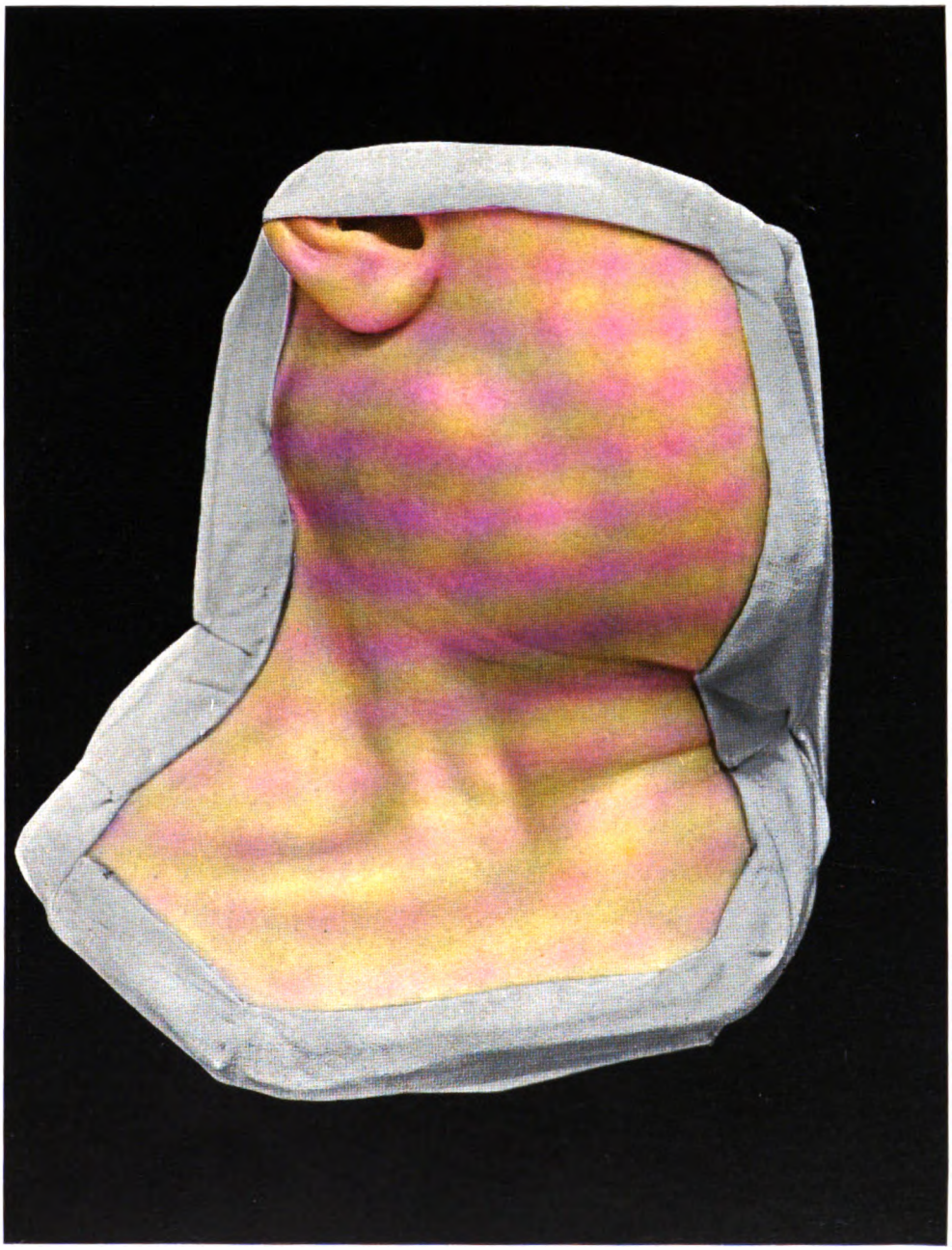

Fig. 124. Lymphomata colli tuberculosa.

Rebman Company New-York.

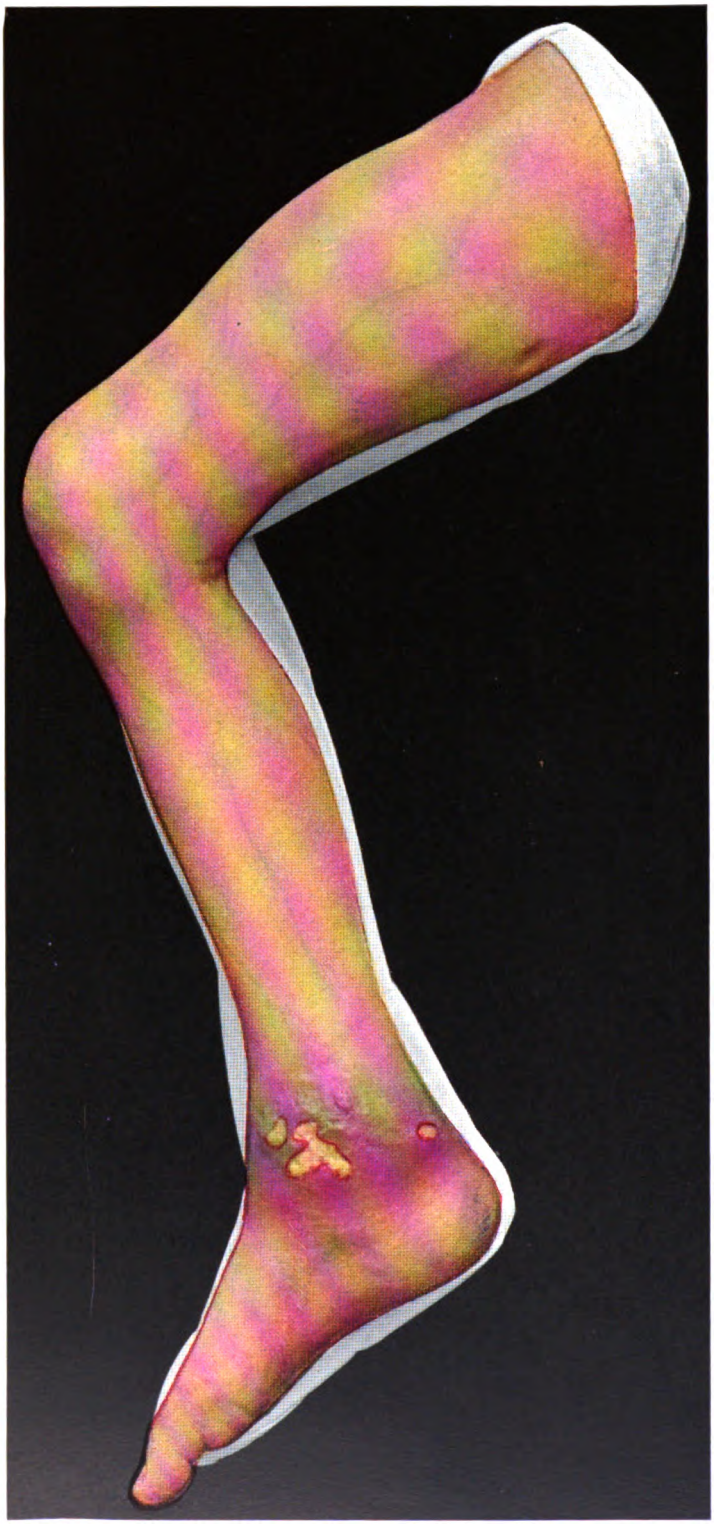

Fig. 125. Arthritis tuberculosa fungosa – Ankylosis genus fibrosa – **Abscessus frigidus.**

Rebman Company, New-York.

ARTHRITIS TUBERCULOSA FUNGOSA
(*Fungoid tuberculous arthritis*)
ANKYLOSIS GENUS FIBROSA (*Fibrous anchylosis*)
ABSCESSUS FRIGIDUS (*Cold Abscess*)
Plate XCIX, Fig. 125.

This is a case of multiple tuberculosis of the joints, bones and soft parts, together with pulmonary tuberculosis, occurring in a young individual. The right leg was useless owing to extensive disease of the hip joint. The thigh was flexed, and X-ray examination showed destruction of the upper margin of the acetabulum and displacement of the head of the femur onto the ilium. In the middle of the flexor surface of the thigh is a healed fistula due to a burrowing abscess. In the middle of the extensor surface of the thigh is a clearly visible swelling due to a burrowing abscess, which is common in this situation in tuberculous arthritis of the hip joint, and in tuberculous disease of the vertebræ; in the latter case the abscess burrows along the psoas muscle. Fluctuation was present, but the skin was intact (cold abscess). The abscess was evacuated by puncture and injected with iodoform-glycerin. Resection of the hip joint was postponed till the general condition of the patient was improved.

The knee joint was also the seat of old tuberculous arthritis of the fibrous type, which had led to anchylosis at right angles. This was corrected under an anæsthetic by forced movement and an extension splint.

On the inner side of the ankle joint are characteristic tuberculous ulcers, with irregular undermined borders and yellow caseous surface. Thin, greenish pus was discharged by pressure. The X-rays showed

a focus of tuberculous disease in the astragalus, which had broken into the joint. Tuberculous arthritis of the ankle joint more often breaks through on the outer side. Hydrops is rare in this situation. The joint is usually filled with fungoid tuberculous tissue which extends to the periarticular tissues. In Fig. 125 the foot was in the position of equinus owing to absence of function and neglect of treatment. Owing to the extensive nature of the disease conservative treatment was out of the question. The joint was freely laid open and all tuberculous matter removed (arthrectomy). The limb was immobilized by plaster of Paris bandages and extension applied.

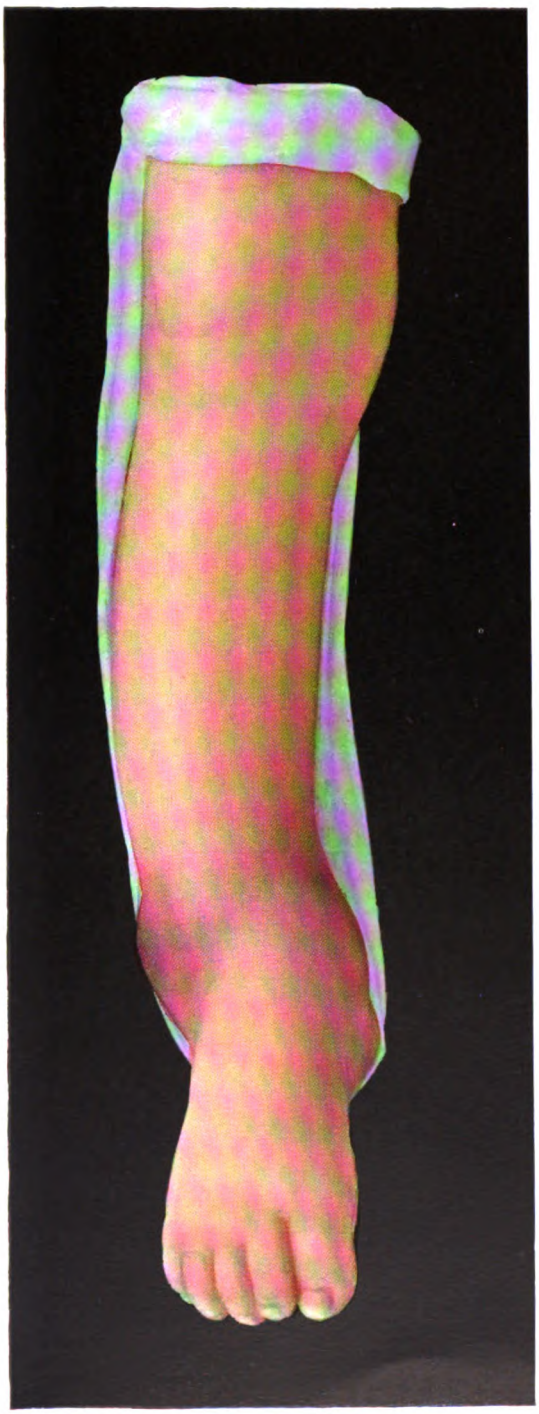

Fig. 126. Arthritis tuberculosa-purulenta.

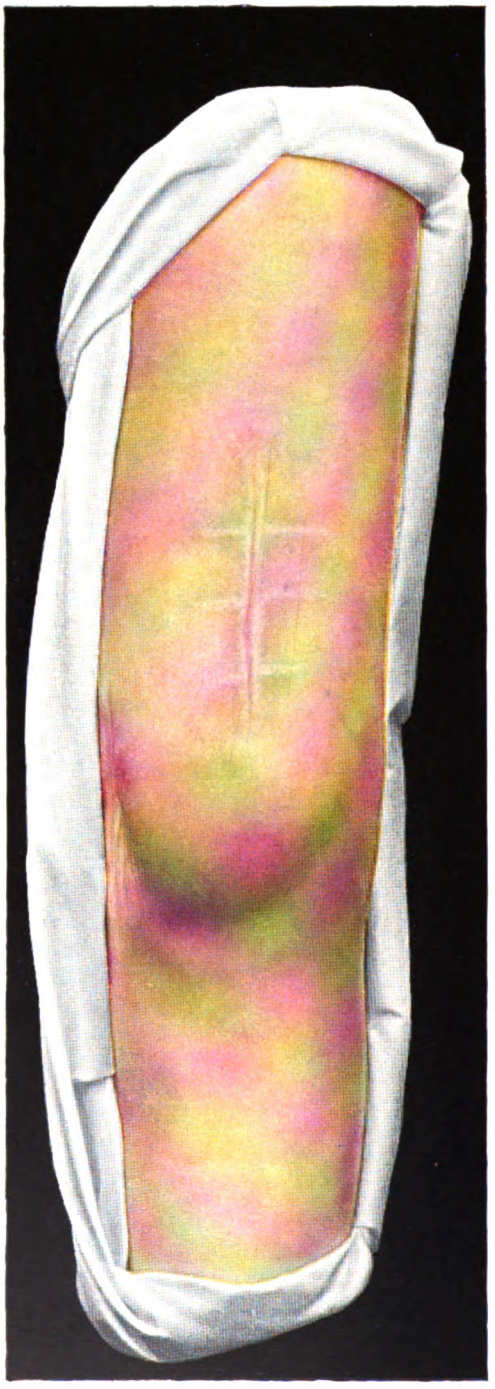

Fig. 127. Arthritis tuberculosa fibrosa – Ankylosis ossea – Subluxatio.

Rebman Company, New-York.

ARTHRITIS TUBERCULOSA PURULENTA

(*Purulent Tuberculous Arthritis*)

Plate C, Fig. 126.

This figure shows a case of purulent tuberculous arthritis of the ankle joint. This form of arthritis is common in children, more often affecting the knee-joint. It begins with fever and rigors, and the rapid formation of abscess, and may be mistaken for arthritis due to staphylococci or other pus cocci. Two incisions were made on the outer and inner sides of the joint, and characteristic thin pus mixed with fibrin was evacuated. The joint was then put up in plaster of Paris. Purulent tuberculous arthritis in children often recovers after early incision; but there is generally some stiffness in the joints, so that these must be put up in the most suitable position for future use.

ARTHRITIS TUBERCULOSA FIBROSA
(*Fibrous Tuberculous Arthritis*)
ANKYLOSIS OSSEA (*Bony Anchylosis*)
SUBLUXATIO (*Sub-luxation*)
Plate C, Fig. 127.

This is a case of old-standing fibrous tuberculous arthritis of the knee joint with bony anchylosis, as shown by the X-rays. Owing to neglect of prolonged fixation of the joint in the straight position, flexion contracture with backward displacement of the tibia has taken place. This was corrected by cuneiform osteotomy, plaster of Paris bandages, and later on a celluloid casing.

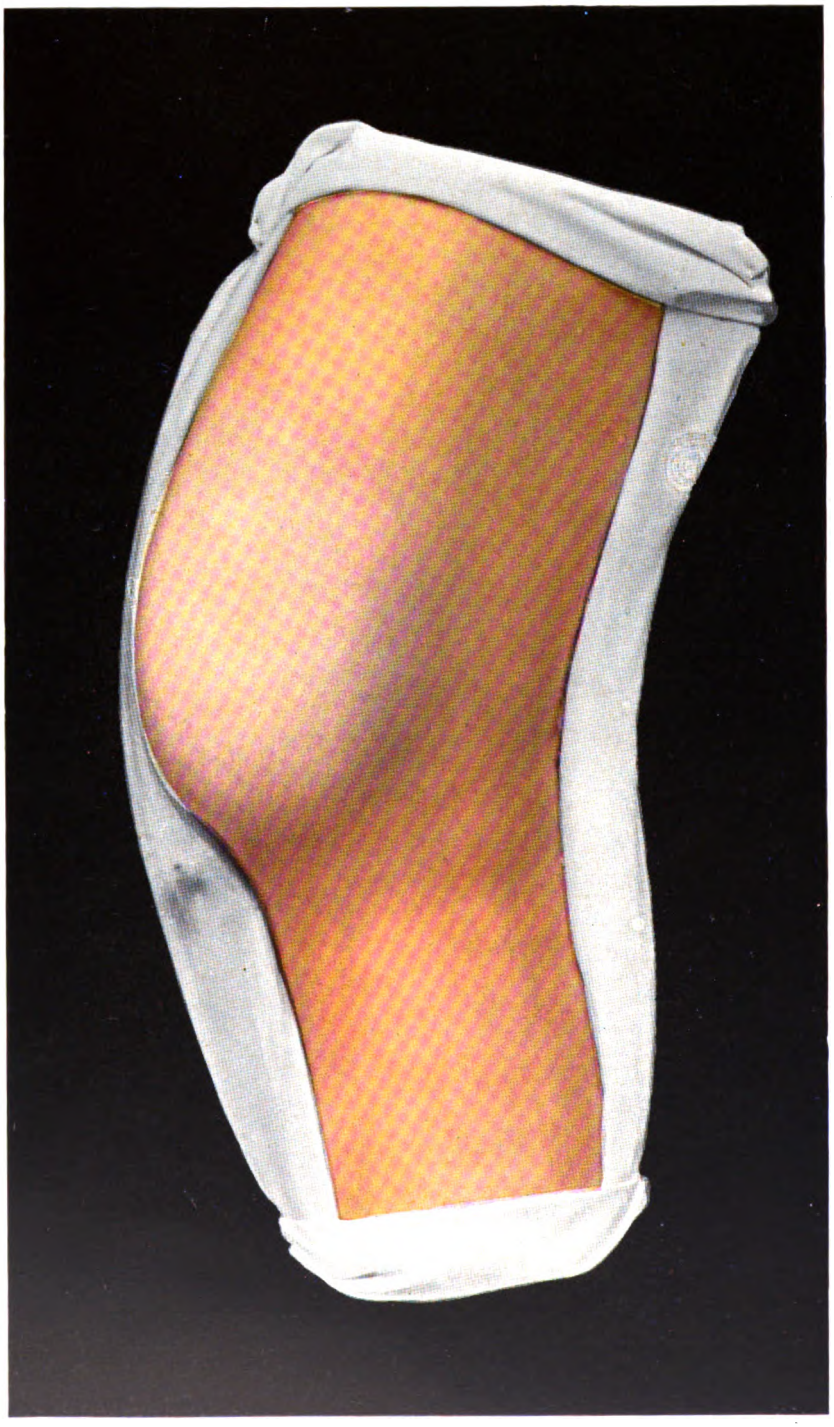

Fig. 128. Arthritis tuberculosa — Tumor albus.

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ARTHRITIS TUBERCULOSA FIBROSA
(Fibrous Tuberculous Arthritis)
TUMOR ALBUS *(White Swelling)*
Plate CI, Fig. 128.

This form of tuberculous arthritis is common in the knee joint in adults. It consists in the formation of hard, fibrous tissue in the joint and periarticular tissue, and gives rise to a tumor-like swelling of the knee and adjacent parts. The skin is white from pressure of the subjacent mass; hence the name white swelling, or *tumor albus*. In Fig. 128 the disease was of several months' duration, and was associated with tuberculosis of the lungs. The patient attributed the affection of the knee to an injury. The X-rays showed tuberculosis of the bones, as well as of the synovial membrane—a common combination in tubercle of the knee joint. Similar swelling occurs in tuberculous hydrops, the simplest form of tuberculous joint disease. Effusion into the joint often precedes the arthritis and is recognized by *balottement* of the patella, which is raised from the femoral condyles by the fluid in the joint. The fluid is generally sero-fibrinous, with numerous free "rice bodies." More common than the fibrous form is fungoid arthritis, which may go on to suppuration and cause much destruction in and around the joint. In all forms of tuberculous arthritis of the knee, the joint is in a position of flexion and valgus. The muscles of the leg become atrophied, and there is retarded growth of the leg.

In Fig. 128, the joint was resected and all tuberculous tissue removed. The articular ends of both

bones were extensively diseased and the cartilages destroyed. In resection of the articular surfaces it is necessary to saw the bones so that the limb can be brought into a straight position.

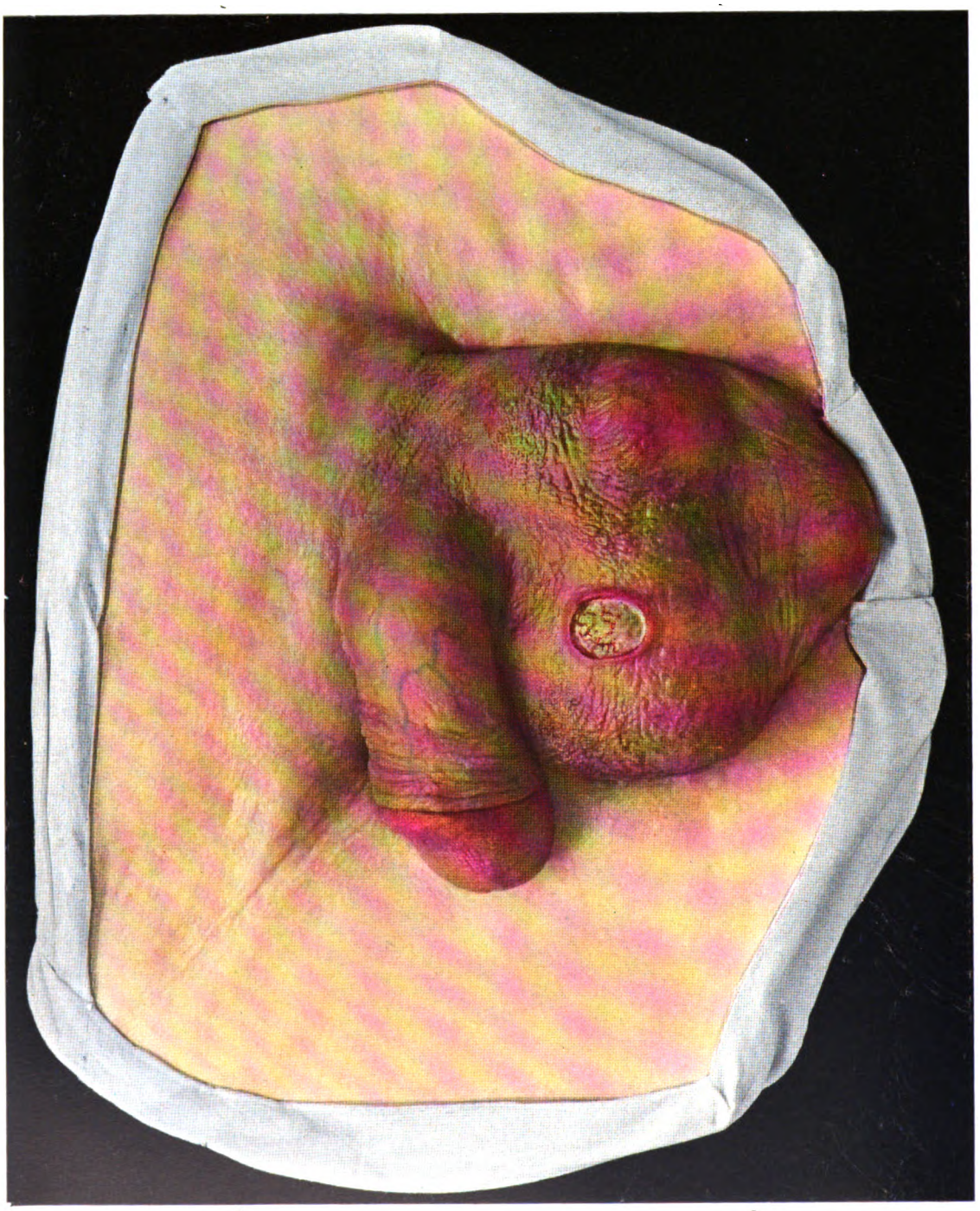

Fig. 129. Tuberculosis testis.

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TUBERCULOSIS TESTIS (*Tuberculosis of the Testicle*)
Plate CII, Fig. 129.

Tuberculosis of the testicle begins in the epididymis and extends to the testicle. It often affects both testicles. There is often tuberculosis of the bladder, kidneys and seminal vesicles, and nearly always pulmonary tuberculosis. In the early stages of the disease hard nodules are felt in the testicle. Later on these nodules become soft and fixed to the skin, which breaks down and forms a typical tuberculous ulcer (Fig. 129). In advanced cases there may be several ulcers and fistulas in the scrotum, discharging caseous pus. The spermatic cord is usually thickened, and the seminal vesicles can sometimes be felt enlarged by rectal examination. The prostate is seldom affected by tuberculosis.

Fig. 129 shows extensive disease of the left testicle and epididymis. The skin is thin in several places, and ulcerated in one place. The spermatic cord was thickened, but no disease was found in the bladder, seminal vesicles, prostate or kidneys. There was advanced tuberculosis of the lungs. In the early stages of the disease the tuberculous foci may be incised and scraped, but more advanced cases require castration (Fig. 129). The testicle when removed showed miliary nodules in some parts, abscesses and caseous foci in other parts.

In its early stage tuberculous testicle may be mistaken for gumma, but the latter begins in the testicle, and takes a long time to break through the skin. Malignant growths cause more rapid enlargement of the testicle.

TUBERCULOSIS MANUS (*Tuberculosis of the Hand*)
Plate CIII, Fig. 130.

In an old woman, who suffered from advanced pulmonary tuberculosis, a swelling gradually developed over the left wrist, causing pain on movement. The swelling gradually extended over the back of the hand, preventing movement of the fingers. Two typical tuberculous ulcers discharging thin pus and caseous matter developed on the back of the hand. Passive movement at the wrist joint was very limited and caused crepitation. The X-rays showed tuberculous disease of the carpal and metacarpal bones. Tuberculosis of the wrist joint in old people is often so extensive as to require amputation. In this case the joint was resected, the cavity filled with iodoform glycerin, and the limb put up in plaster of Paris.

The operation showed the presence of tuberculosis of the tendon-sheaths (tendovaginitis), the tendons being imbedded in granulation tissue. Tuberculous tendovaginitis is more common in the upper extremity, and occurs apart from bone disease. It may take the form of tuberculous hygroma, with serofibrinous fluid and crepitation on movement of the tendons; or a fungoid form in which the tendons are imbedded in spongy granulations. Tuberculous disease of the tendon-sheaths is most extensive when it is secondary to old-standing tuberculous joint disease, as in the above case. The treatment consists in removing the diseased tissue without injuring the tendons; a difficult operation in the case of flexor tendons, on account of the vessels and nerves.

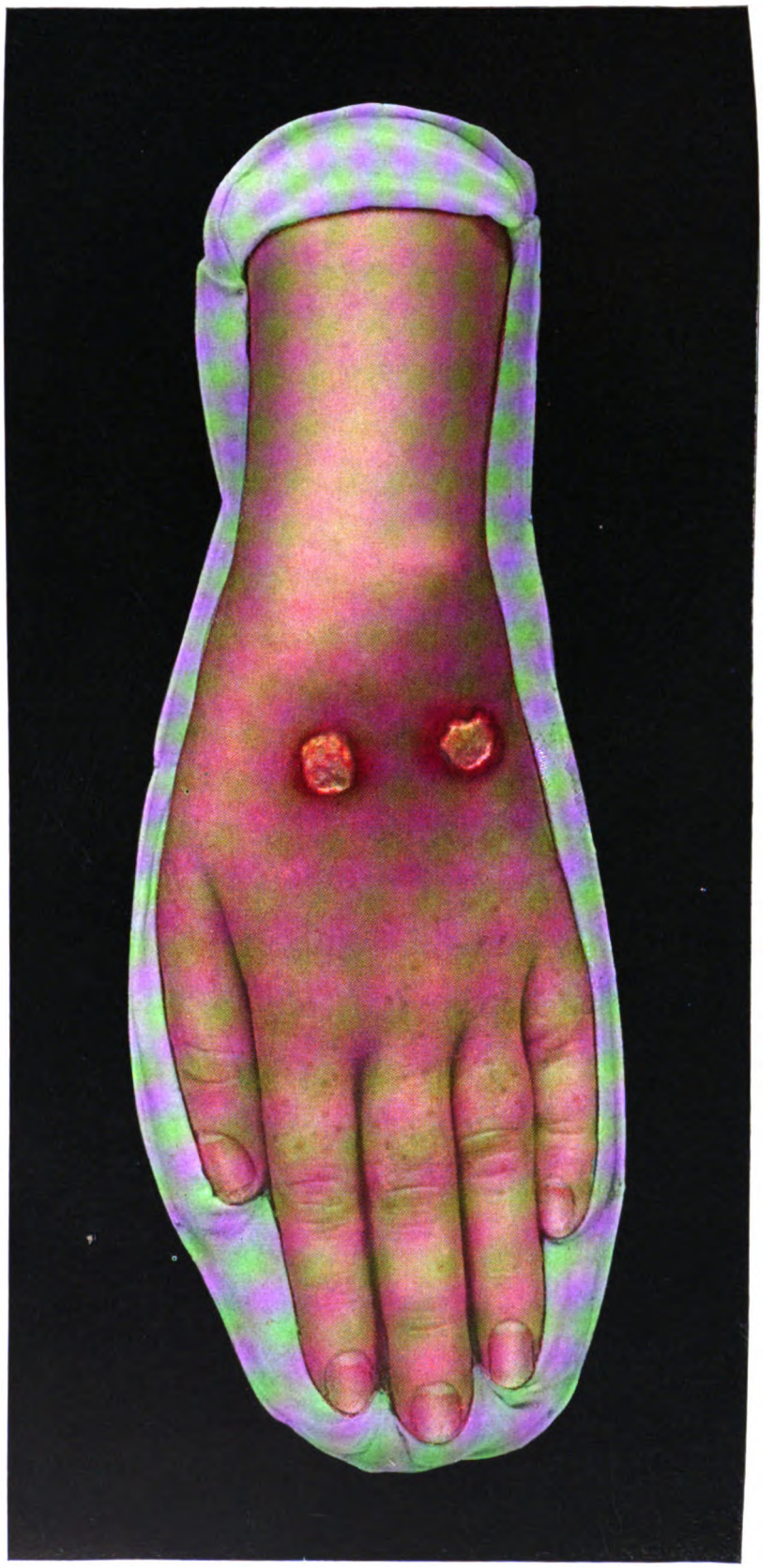

Fig. 130. Tuberculosis manus.

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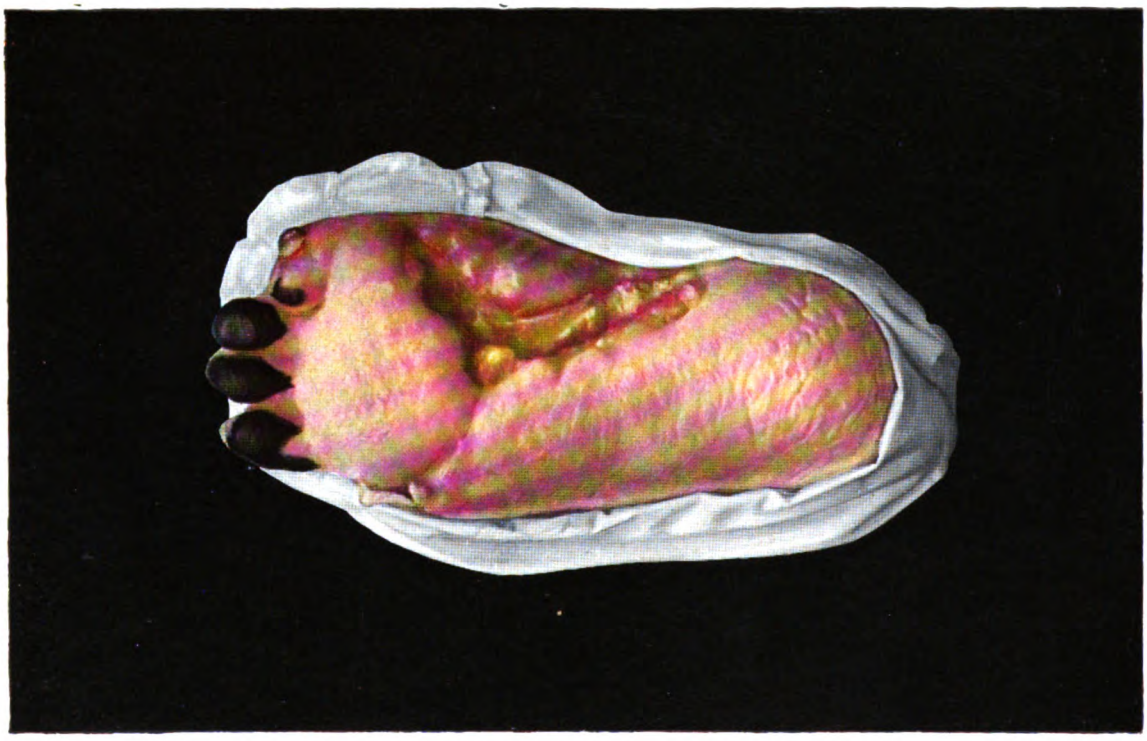

Fig. 132. Gangraena humida pedis.

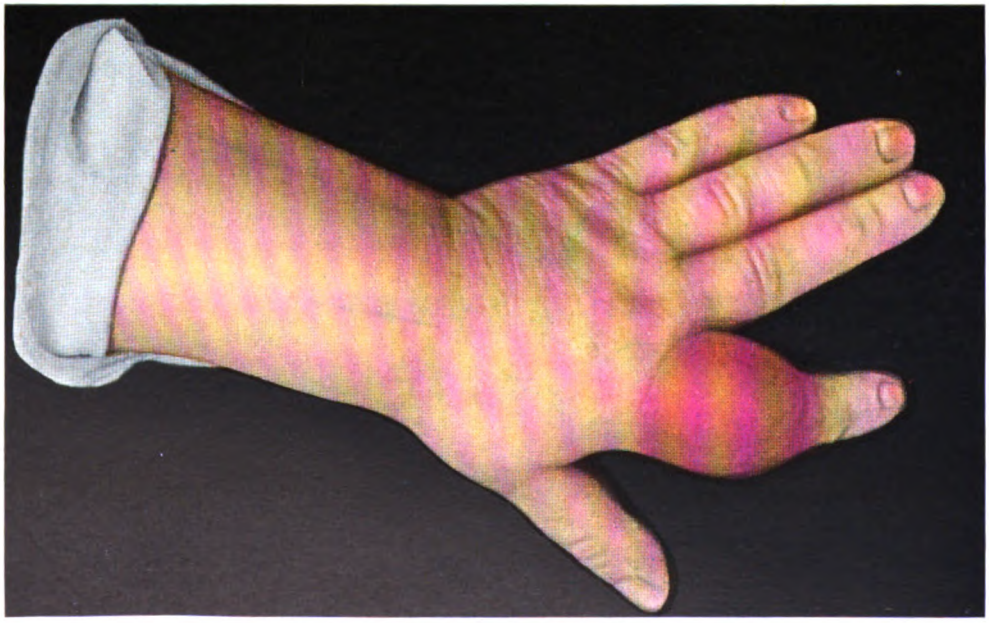

Fig. 131. Ostitis tuberculosa — Spina ventosa.

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OSTITIS TUBERCULOSA (*Tuberculous Osteitis*)
SPINA VENTOSA (*Dactylitis*)
Plate CIV, Fig. 131.

Tuberculosis of the phalanges begins in the medulla and extends to the cortex and periosteum. The whole diaphysis may be destroyed by suppuration and caseation, while the periosteum forms a thin shell of new bone. The bone then appears swollen, as if inflated (spina ventosa). The disease generally affects several phalanges of several fingers on both hands, and is often found in the children of tuberculous parents. The destructive process is more severe than in any other form of tuberculous osteitis, several phalanges being often completely destroyed. Fistulas form in the œdematous skin and discharge caseous matter. Growth of the fingers is interfered with, so that they often form deformed stumps after the disease has healed. The disease is often overlooked as it is at first painless; but early diagnosis can be made by the X-rays which show the changes in the bone.

Syphilitic dactylitis differs in causing less destruction of bone, and in the usual absence of suppuration and necrosis; but the diagnosis often depends on other signs and history of syphilis or tuberculosis.

Treatment consists in early incision, scraping, and plugging with iodoform gauze.

GANGRÆNA PEDIS HUMIDA (*Moist Gangrene of the Foot*)
Plate CIV, Fig. 132.

This case is of special interest, gangrene of the foot having developed after forcible correction of flexion contracture due to tuberculosis of the hip joint. Soon after this operation the toes became cold, blue and flexed, and finally black. As the gangrene was limited to the anterior portion of the foot, it is probable that the injury was to the intima only and not a complete rupture of the femoral artery, and that gangrene was due to thrombosis of the vessel.

The figure shows gangrene gradually involving the anterior part of the foot. In the first and fifth toes necrotic bone emerges from fistulas in the skin. In the sole of the foot a wide zone of demarcation is seen, covered with granulations, and separating the gangrenous part from the healthy tissues behind. When the line of demarcation has extended all round the foot, the gangrenous part can be removed, and the wound can be repaired by an osteoplastic operation. The different forms of gangrene will be described with the next plate.

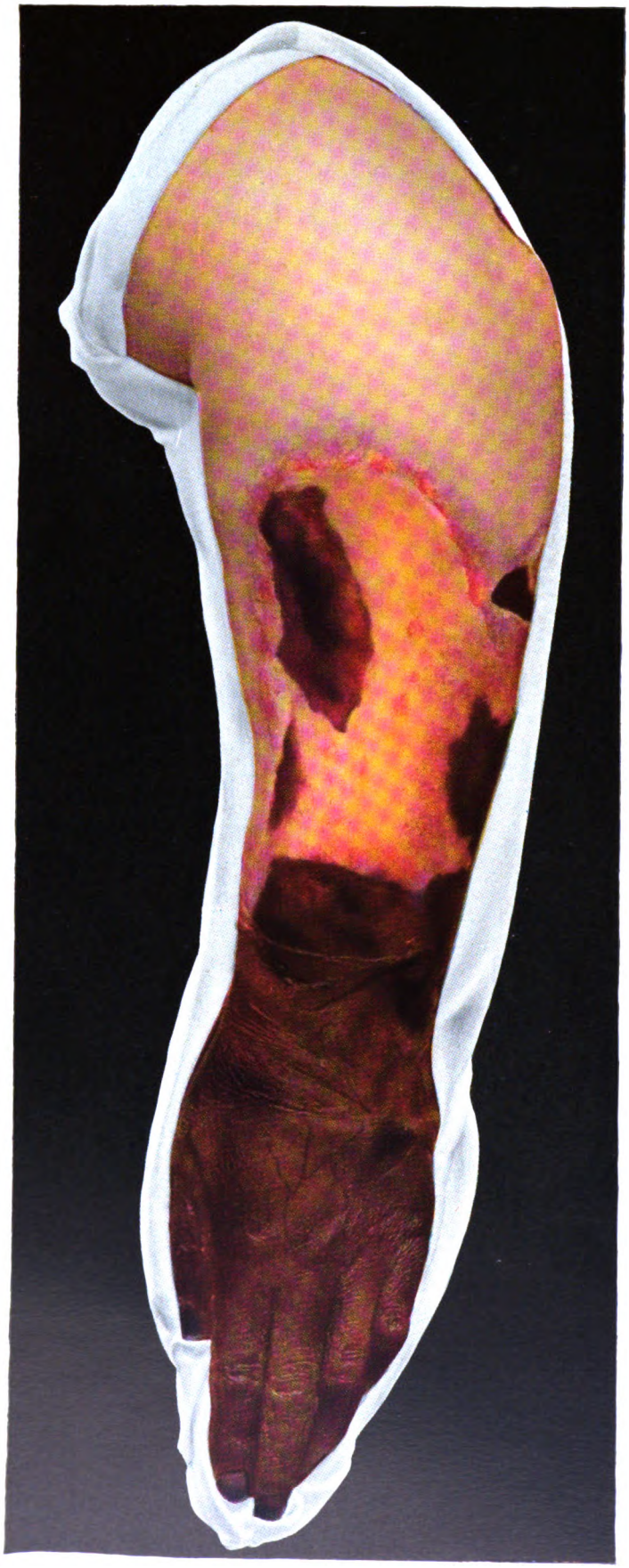

Fig. 133. Gangraena sicca brachii — Mumificatio.

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GANGRÆNA SICCA BRACHII—MUMMIFICATIO

(*Dry Gangrene of the Arm*)—(*Mummification*)

Plate CV, Fig. 133.

The term gangrene is applied to extensive, progressive death of the superficial tissues of the body; the term necrosis to death of the deeper structures (fascia, muscle and bone). The bones have a greater power of resistance than the skin, which may become gangrenous after slight disturbance in the circulation. Under certain conditions, *e.g.* after cutting off the blood supply, the whole peripheral part of a limb may become destroyed; but, as the death of the tissues is first noticed in the skin, it is spoken of as gangrene. When the process consists in desiccation of the tissues it is called dry gangrene; when it ends in liquefaction from the invasion of putrefactive bacteria, it is called moist or infective gangrene. Dry gangrene may change to moist gangrene, and both processes may occur simultaneously in different parts of the same limb, when one part becomes infected and the other does not.

The extent of the gangrene varies according to the cause; it may be circumscribed (after local applications, such as carbolic acid), or progressive (after embolism). In both forms the dead tissue becomes separated from the living by a zone of demarcation. The zone of demarcation forms a groove filled with granulation tissue (Fig. 132). It may be circular (Fig. 135) or irregular (Figs. 133 and 134).

In the early stage of dry gangrene the condition resembles that of ischæmic muscular contracture (Fig. 63), especially when the condition is due to plugging of the blood-vessels. The skin becomes

dry, shrunken and parchment-like. In the extremities the peripheral parts are flexed and immovable. The skin becomes gradually yellowish brown and finally black (Fig. 133). All the subjacent structures may undergo dry atrophy. The dead tissue is gradually separated by the zone of demarcation, and the whole of an extremity may undergo spontaneous separation.

While in dry gangrene there is diminution in volume and charring of the affected part; in moist gangrene there is increase in volume, due to preceding œdema. In moist gangrene there is more or less liquefaction, decomposition or putrefaction, due to putrefactive bacteria. The skin is cool and moist, and the epidermis becomes raised in bullæ containing blood-stained fluid. After rupture of the bullæ the skin is reddish brown (Fig. 109). Finally the tissues become disintegrated and smell horribly; lymphangitis, lymphadenitis and general infection then follow.

In both forms of gangrene the skin is at first pale and cold, and then shows bluish patches in various places, often without any direct connection. Contractures and loss of movement indicate the occurrence of total gangrene, whether moist or dry. However, the difference in volume between the two forms is apparent from the beginning.

A deep groove of demarcation also forms in moist gangrene, separating the dead from the living tissue, and spontaneous separation may occur if the patient does not succumb to general infection. In less extensive cases of moist gangrene we can wait for the line of demarcation to form; but the gangrenous part must be removed if there are rigors and high temperature.

The etiology of gangrene is complex, but it is always due to disturbance of the circulation. The blood-vessels may be affected directly or indirectly. Senile gangrene in old people is due to arteriosclerosis. The loosened intima of the small terminal

vessels (also in the larger vessels) gives rise to thrombosis, causing death of the peripheral tissues supplied by these vessels, especially when the vessels which carry on collateral circulation are themselves diseased. In this way the toes or the whole leg may become gangrenous. In these cases the typical changes of gangrene are preceded by pain. Gangrene of the lower extremities in diabetic subjects is generally caused by disease of the vessels.

In younger people gangrene of the peripheral parts of the extremities may be caused by disease of the intima of the smaller vessels (endarteritis obliterans). This is usually of syphilitic origin. In these cases there are severe intermittent pains, causing the patient to limp (intermittent claudication). Both feet are usually affected, and become bluish red. After some years gangrene gradually supervenes, often taking months to develop (angiosclerotic gangrene). The patients suffer severe pain, especially on contact or exposure to cold.

Embolism of the main arteries (*e.g.* from heart disease) causes sudden and extensive gangrene of the upper or lower extremities. (Embolic gangrene). Sudden gangrene may also be caused by rupture or ligation of a main artery. Certain nervous diseases may cause gangrene by vaso-motor constriction of the vessels (angio-neurotic gangrene). The latter affection occurs symmetrically in both feet and is known in its early stages as *Raynaud's* disease. It is generally preceded by paræsthesias and diminution in the sense of temperature.

Gangrene may also occur after extensive burns and frostbite; after local application of carbolic acid, lysol and alcohol; after injection of adrenalin into the tissues, and after the internal administration of ergotin (hands, feet and ears). In all these cases gangrene is caused by thrombosis of the vessels. In the same way erysipelas and phlegmonous inflammation may cause gangrene of the skin and deeper

tissues. Gangrene of the skin may also be caused by the X-rays and by radium.

Differential Diagnosis. The appearance of gangrene, when fully developed, is so characteristic that it can hardly be mistaken for any other condition. The two forms of gangrene are also sharply defined from each other. Dry gangrene might be mistaken for burns of the third or fourth degrees, if signs of the first and second degree of burn were not always present in the neighborhood. Moist gangrene might be mistaken for putrefactive phlegmon, especially with progressive gaseous phlegmon (Fig. 109), if the signs of general infection were not present at an early stage. The history and a thorough examination will not only establish the diagnosis, but in most cases will decide the cause of the gangrene.

The prognosis naturally depends on the cause and on the extent of the gangrene. Angiosclerotic gangrene extends very slowly; it may remain stationary; or parts which appeared to be affected may recover. Plugging of a large vessel causes extensive gangrene of the part supplied by the vessel. Diabetic gangrene and senile gangrene are characterized by their progressive course. Gangrene is more extensive when there is much œdema.

Treatment. Extensive gangrenous parts should be removed after a zone of demarcation has formed. Before this takes place the part should be dressed with aseptic dressings or ointments. In moist gangrene of an extremity early removal may be indicated in order to prevent general infection. In gangrene due to syphilitic endarteritis, iodide of potassium and mercury should be given; the limb should be raised and enveloped in wool; hot-air treatment is useful for the pains; alcohol should be avoided: after demarcation has formed, amputation should be performed in the most conservative way possible.

In embolic and in diabetic gangrene high amputation is often necessary.

In amputation the elastic tourniquet is to be omitted in cases where the gangrene is due to changes in the vessels, as it may cause further gangrene above the point of amputation. If the vessels in the stump only bleed slightly, this shows that they are already affected and that the gangrene will probably extend further. The veins in the amputation-stump bleed freely, owing to the absence of the *vis a tergo* due to narrowing of the arteries. After amputation any pressure of the dressings is to be avoided.

Fig. 132 shows a typical case of dry gangrene or mummification of the arm, affecting all the tissues. The fingers are contracted and blackish brown in color. The skin is hard. In the forearm commencing gangrene is seen in the yellow leathery skin. The line of demarcation is seen as a red zone formed of granulation tissue, separating the dead from the healthy parts. After the line of demarcation had extended all round the limb, amputation through the arm was performed.

In this case gangrene was due to rupture of the axillary artery during an operation for reduction of an old dislocation. In old dislocations at the shoulder joint bloodless reduction is generally impossible and may cause rupture of the artery. But this disadvantage also applies to reduction by open operation, for the displaced vessels are liable to become damaged by pressure of the dislocated head of the humerus and are easily ruptured during reduction of the dislocation. This accident may be avoided by resection of the head of the humerus, after carefully separating the artery, which is generally united to it. The incision for the operation is the same as for ligation of the axillary artery.

This case also shows the importance of early diagnosis of dislocation of the humerus, which is easily made by the X-rays.

GANGRÆNA CUTIS HUMIDA—NECROSIS FASCIÆ

(*Moist Gangrene of the Skin*)

ULCUS DECUBITALE (*Decubital Ulcer—Bedsore*)

Plate CVI, Fig. 134.

The skin, being the most superficial part of the body, is most liable to injuries which may cause gangrene. It has also less power of resistance than other tissues. Long-continued pressure, especially in places situated over the bones, may cause gangrene of the skin. In this way gangrene may be caused by the pressure of tight bandages or splints; also by a displaced piece of bone in fractures; by pressure on the outer side of the foot in pes varus; by tight sutures, *e.g.* after amputation of the breast, leaving a wide space to be closed.

Uncleanliness, loss of consciousness, nervous diseases (trophoneuroses, syringomyelia, hemiplegia, paraplegia, tabes), cachexia, diabetes, typhoid fever, osteomyelitis, phlegmonous inflammation, general infection and comatose conditions, all predispose to gangrene, which, in emaciated persons, may become very extensive. Gangrene of the skin caused by the pressure of œdema and gaseous formation in the tissues has already been mentioned (Figs. 91 and 109). After operations, gangrene of the skin (bedsores) may occur over the heels, buttocks, spinous processes, shoulder blades and back of the head, if care is not taken to change the position of the patient and apply soft, smooth, protective coverings.

Gangrene of the mucous membranes may occur from the pressure of foreign bodies; for instance, in the esophagus, from the passage of bougies; in the intestine, from the pressure of *Murphy's* buttons; in

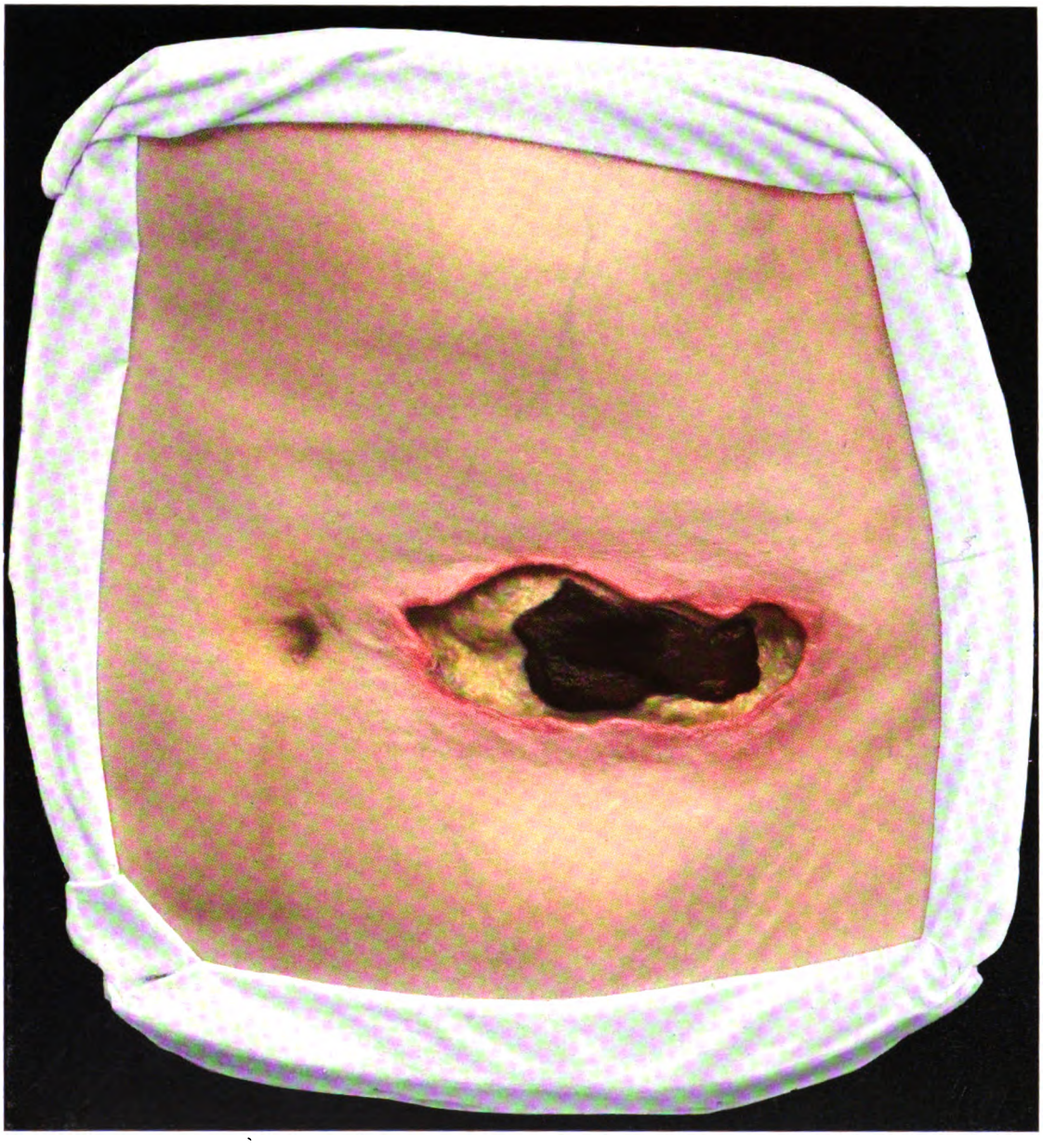

Fig. 134. Gangraena humida cutis – Necrosis fasciae – Ulcus decubitale.

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the larynx, after intubation; also after resection of the intestine or esophagus when the united ends are under great tension.

Gangrene of the skin begins with pain and redness; then slight swelling and blue coloration; finally, raising of the epidermis in bullæ. The epidermis then separates leaving the corium exposed; this is at first greenish yellow, afterwards blackish brown and leathery. At the edge of the gangrenous part the skin becomes inflamed, and by the formation of pus and granulation tissue a gutter-shaped, often circular space is gradually formed—the zone of demarcation. The more severe the injury the deeper is the gangrene so that subcutaneous tissue, fascia (Fig. 134), muscles and bone may become necrosed and cast off.

After separation of the gangrenous part an ulcer is left, called decubital ulcer, which is covered with slimy, greenish-yellow connective-tissue shreds and fetid pus. A neglected decubital ulcer may give rise to extensive putrid inflammation or gaseous phlegmon, as the pus always contains putrefactive bacteria, especially in decubital ulcer over the sacrum which is infected from the fæces. Erysipelas may also occur in decubital ulcer. In neglected cases the gangrene may also extend deeply and cause extensive destruction.

Pressure-necrosis in the internal organs (larynx, esophagus, intestine) is dangerous from perforating ulceration or hemorrhage; also from stenosis after healing.

Treatment. Gangrene of the skin may, in many cases, be prevented, or, at any rate limited, by prophylactic treatment. Decubital ulcers (bedsores) may be prevented by applications of spirit of camphor to the skin of the parts exposed to pressure, by air cushions and frequently changing the patient's position. If the skin is discolored an ointment dressing should be applied, and this should be changed if

the patient complains of pain. As the pain also subsides in a few days under continuous pressure of a dressing, its removal is often neglected, and then when it is removed there may be gangrene down to the bone. In emaciated patients the bony prominences should, therefore, be well padded, and the skin disinfected before applying the dressing.

If gangrene has developed the skin must be protected against infection by a dressing. Separation of the gangrenous part may be hastened by moist dressings with two per cent. boric acid lotion, three per cent. peroxide lotion, or camphor liniment, applied several times daily. Forcible removal of the gangrenous parts while they are firmly attached is not advisable; they should be removed by scissors when almost completely loose. The ulcer may be treated with moist dressings or ointments, and with caustics when granulations have sprung up. After extensive gangrene of the skin the space may be closed by undermining the skin and suturing; or, if this is impossible, by a plastic operation by means of pedunculated flaps.

Fig. 134 shows a case of moist gangrene of the skin with necrosis of the abdominal fascia. Part of the skin is separated from the healthy, somewhat reddened and inflamed skin around it, by a zone of demarcation. The gangrenous part is still firmly attached to the subjacent structures. In some places the skin has separated, exposing the abdominal fascia, the yellowish color of which shows that it has already undergone necrosis. The borders of the ulcer were undermined, and it discharged fetid pus.

In this case the gangrene was caused by a subcutaneous injection of salt solution, performed on a patient in a state of collapse. Gangrene of the skin may occur after injection of large quantities of salt solution when the injection is made intracutaneously

instead of subcutaneously; also when the fluid is too hot, or not sterilized.

The ulcer became clean under dressings of peroxide lotion; the gangrenous skin and necrotic fascia separated; the edges of the fascia and the skin were sutured separately, and primary union took place. As sutures in fascia often do not hold, the patient was ordered an abdominal belt to prevent abdominal hernia.

GANGRÆNA CARBOLICA (*Carbolic gangrene*)
Plate CVII, Fig. 135.

In this case carbolic acid dressings were applied to a wound in the finger. The end of the finger became white and the epidermis was destroyed as far as the carbolic acid dressing extended, exposing the corium. The patient had no feeling in the tip of the finger and suffered from severe pain. The tip of the finger gradually became black and shrunken (dry gangrene).

The figure shows gangrene of the terminal phalanx. The greenish-yellow color at the junction of the terminal with the middle phalanx indicates commencing gangrene. In the middle of the second phalanx there is a wide zone of granulation tissue indicating the line of demarcation. Severe pain in the finger was due to thrombosis of the terminal arteries caused by the action of carbolic acid. Later on there was loss of sensation in the finger from paralysis of the sensory nerves.

Moist dressings were applied, and in a few weeks a groove of demarcation extended down to the bone. In the peripheral part gangrene extended to the fascia, muscles, tendons and bone. Healing took place after disarticulation at the interphalangeal joint.

It must be borne in mind that even one per cent. carbolic lotion, after a few hours' application only, may cause gangrene of the skin and deep necrosis by thrombosis of the vessels. Certain individuals appear to be predisposed to gangrene after fomentations with carbolic acid, and sometimes lysol or alcohol; especially when gutta percha tissue is placed over them, preventing evaporation. After a short application the skin may recover. Acetic acid dressings hasten recovery.

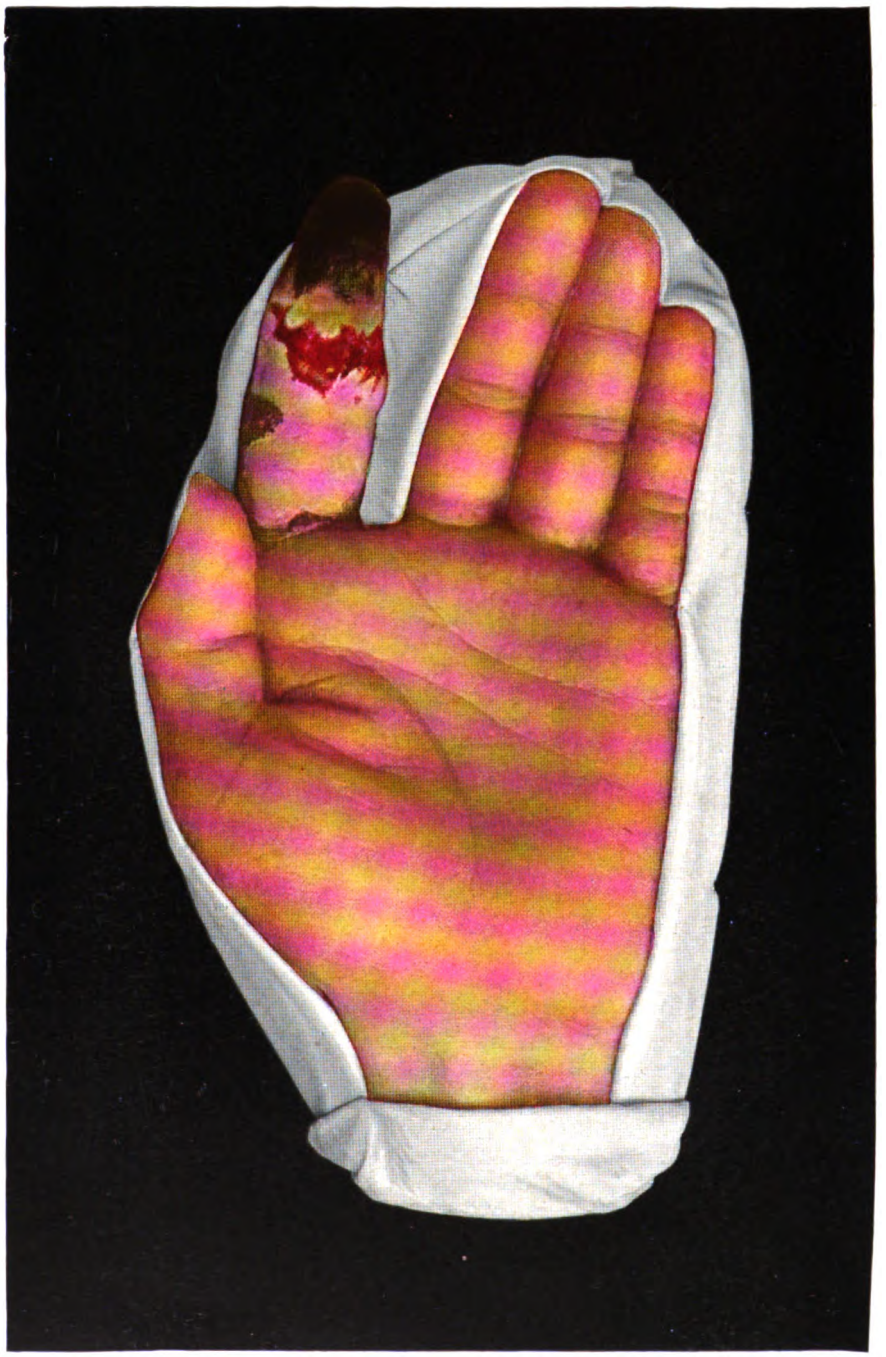

Fig. 135. Gangraena carbolica.

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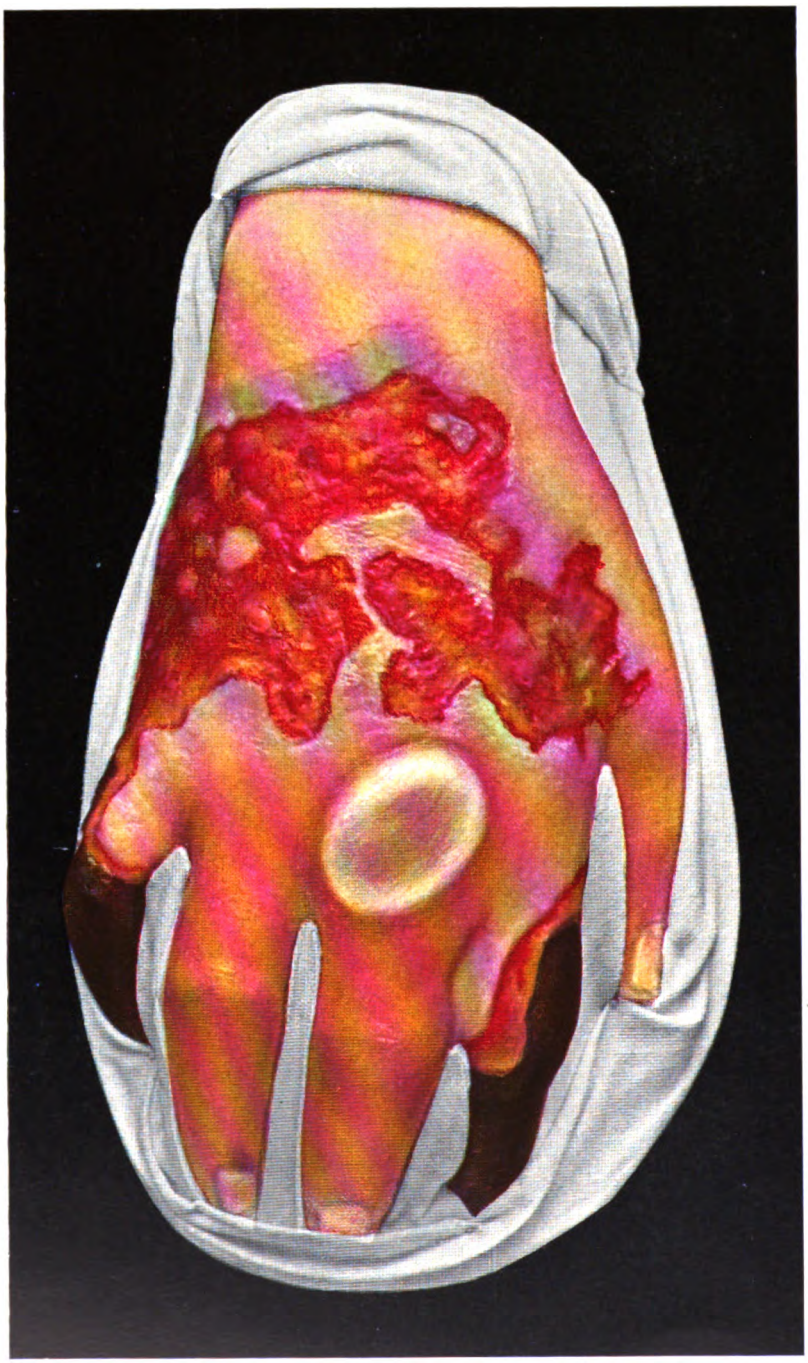

Fig. 136. Combustio erythematosa – bullosa – escharotica.

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COMBUSTIO ERYTHEMATOSA—BULLOSA—ESCHAROTICA

(*Burns*)

Plate CVIII, Fig. 136.

Burns may be caused by the action of radiant heat; *e.g.* prolonged exposure to hot sun or a hot fire. The heat may arise from solids, liquids or gases. Electricity (lightning stroke) and the X-rays may also cause burns; also strong acids and alkalis (sulphuric and nitric acids, caustic potash and soda).

Burns of the mucous membrane of the mouth, tongue, pharynx, esophagus and intestine are caused by certain chemicals swallowed as poisons. These may cause death by œdema of the glottis, or later on by perforating ulceration of the gut and peritonitis. If the ulcers heal, they lead to stenosis of the gut.

Burns of the skin may be caused by strong caustics; such as trichloracetic acid, for removal of warts; Vienna paste, tartar emetic ointment, etc.

Tender skins (*e.g.* children) react to slight degrees of heat; *e.g.* after the application of poultices, fomentations.

The mildest degree of burn—also called the first degree—consists in arterial hyperæmia, causing redness and slight swelling of the skin. There is more or less pain or tenderness, itching and tension of the skin. In this form there is early and complete restitution to normal, sometimes after desquamation of the epidermis.

The second degree of burn is characterized by the formation of bullæ. Besides redness caused by the first degree, the epidermis is raised in blisters by exudation of lymph between the epidermis and the corium. The blisters contain yellowish fluid or gelatinous masses, and may develop twenty-four

hours after the injury. In severe burns of the second degree some of the blisters rupture, exposing the red corium, which is very painful to touch. This form of burn is common after boiler explosions, gas explosions, and scalding with steam or hot water. Healing by epidermization of the corium takes two or three weeks, but the skin is restored to the normal condition without scarring, provided the process has not been complicated by suppuration.

In the third degree of burn the epidermis and corium are destroyed, to a greater or less extent according to the severity of the injury. The resulting gangrene of the tissues is due to three factors; loss of water from the tissues; loss of blood supply from acute thrombosis of the vessels; and coagulation of albumen in the tissues. The skin becomes black and gangrenous. Sensation is lost at this part; but there is always pain due to burns of the first and second degrees in the surrounding parts.

In severe burns the fascia, muscles and bones may undergo necrosis, as well as the skin. The separation of the necrosed parts takes place in the usual way by an inflammatory zone of demarcation. Burns of the third degree are liable to infection of the exposed tissues by pyogenic and putrefactive bacteria, so that the wounds take months to heal, with hypertrophic scars which cause contractures of the joints and form adhesions with neighboring parts. The scars of burns of the third degree are easily lacerated and may give rise to carcinoma (Fig. 20). In burns of the third degree there is also the danger of general infection from prolonged suppuration. These cases may also be fatal from exhaustion, hemorrhage from erosion of vessels, or amyloid disease of the kidney, liver, etc.

In burns of the fourth degree there is complete charring of all the tissues, which fall to ashes when touched.

It is obvious that the effect of a burn on the organ-

ism depends on the degree of the burn, the extent of surface involved, the part of the body affected, and the condition of the patient beforehand. Burns of the first and second degrees when they are not very extensive are not serious; but if a third of the body is affected, even in burns of the first degree, there is fatal constitutional disturbance, especially in children. Apart from the severe pain, rapid collapse sets in. The skin becomes cold, pale and covered with sweat; the pulse is small and rapid; the patient complains of thirst; consciousness is retained till death occurs in two or three days. As the patients are fully conscious and in good spirits, and do not complain of any more pain, it is necessary to explain to the relatives and friends that death after extensive burns is almost inevitable.

In these severe cases the temperature is subnormal. There is sometimes delirium and coma. In extensive burns of the second degree, complicated by general infection, there is high temperature, delirium, diarrhea and fetid discharge from the wounds. Duodenal ulcer may also occur. Death may occur from uræmic coma following anuria. The autopsy shows ecchymoses and thromboses in all the organs, parenchymatous nephritis, etc.

In extensive burns death may be caused by shock, which may be due to great pain, sudden cooling of the skin, or overheating of the blood, as in heat-stroke. Accumulation of poisonous substances in the blood may also cause death.

Burns of the third degree, when affecting certain regions, give rise to various disfigurements and contractures. The eyelids and mouth may be disfigured by contracting scars (ectropion). The head may be flexed on the thorax; the fingers may become united, etc.

Differential Diagnosis. Burns may be confounded with frostbite, in the absence of history.

Treatment. In burns of the first degree the surrounding healthy skin should be disinfected, and ointment applied to the burnt part. In burns of the second degree small blisters can be left to dry up; large blisters should be opened at the base, the lymph evacuated and the epidermis replaced. The loosened epidermis then generally becomes attached. If the blisters are already broken, the loose epidermis should be removed and the exposed corium powdered with rice powder, talc or flour. Bismuth dressings are useful; but oil and lime water applications should not be employed, as they favor infection. Antiseptic gauze is to be avoided, on account of the danger of poisoning.

Morphia may be required if there is much pain, especially when the dressings are changed. The latter should be covered with plenty of wool.

The more infection of the surface is prevented by careful treatment, the less is the scar tissue. If there is much scar tissue this may be excised and the wound covered by skin flaps. Injection of ten per cent. thiosinamin solution may be tried to absorb scar tissue.

In the extremities, resection of joints, amputation or disarticulation may be necessary when the limbs are useless, or the seat of exhausting suppuration, or when there is threatening general infection. Such operations should not be performed till the patient has somewhat recovered from shock. Burns of the neck and mouth may require tracheotomy. In extensive burns of the second and third degrees with much discharge permanent baths are useful.

In all severe burns the general condition of the patient requires attention. To support the heart, digitalis, camphor injections, subcutaneous injections of salt solution may be indicated. The whole body must be well protected by wool. The function of the kidneys should be stimulated by diuretics (caffein, acetate of potash, etc.).

Burns caused by acids require neutralization by the application of alkalis (*e.g.* soap); while burns caused by alkalis require neutralization by weak acids (acetic acid, vinegar). Internal burns caused by swallowing chemicals may require special surgical treatment for the resulting stenosis.

In lightning-stroke treatment is generally useless. In heat-stroke and sunstroke, the overheated body must be cooled by applying ice bags to the head and over the heart, and by drinking large quantities of water.

Fig. 136 shows all four degrees of burns. In this case the injury was caused by red-hot metal. The first degree is shown by reddening of the epidermis; the second degree by the formation of blisters containing yellow fluid; the third degree (on the back of the hand) by the destruction of epidermis, exposing the corium, and in some places the bones; the fourth degree by charring of the ends of the second and fifth fingers. The first phalanges of these fingers also show burns of the third degree. The different degrees of burn are due to the differences in the length of time during which the heat was acting in the different places. The second and fifth fingers were disarticulated; the rest of the hand recovered, with moderate function, after treatment by the permanent water bath.

CONGELATIO ERYTHEMATOSA—BULLOSA (*Frostbite*)

Plate CIX, Fig. 137

Extreme degrees of cold may cause destruction of the tissues, in the same way as burns. Here again, the extent of injury depends on the degree of cold, the duration of its action, and the condition of the patient. Dry cold is better borne than moist cold. Certain individuals are especially liable to the effects of cold—persons in a state of alcoholic intoxication, anæmic individuals, children and old people, cooks and others who are exposed to rapid changes of temperature. Frostbite may be caused by the action of snow, ice or liquid air.

Pernio or chilblain may be regarded as a chronic form of frostbite, affecting the fingers, toes and ears. It is especially common in chlorotic individuals and causes swelling and blueness of the skin with numerous bluish-red nodules. These often cause unbearable itching and burning sensations, and, when scratched, give rise to intractable ulcers.

Acute frostbite appears in different degrees according to the degree of cold, in the same way as burns. The parts of the body usually affected are the fingers, ears, nose and toes. In the first degree of frostbite there is redness of the skin from hyperæmia (erythematous congelation). This is usually followed in a short time by the formation of a blister. The redness increases when the patient comes into a warm room, or takes alcoholic drinks. It is accompanied by burning and itching pains, which may continue for a long time. The redness may even last for life after a single frostbite of the first degree; for instance in the nose of a chlorotic woman. In most cases of

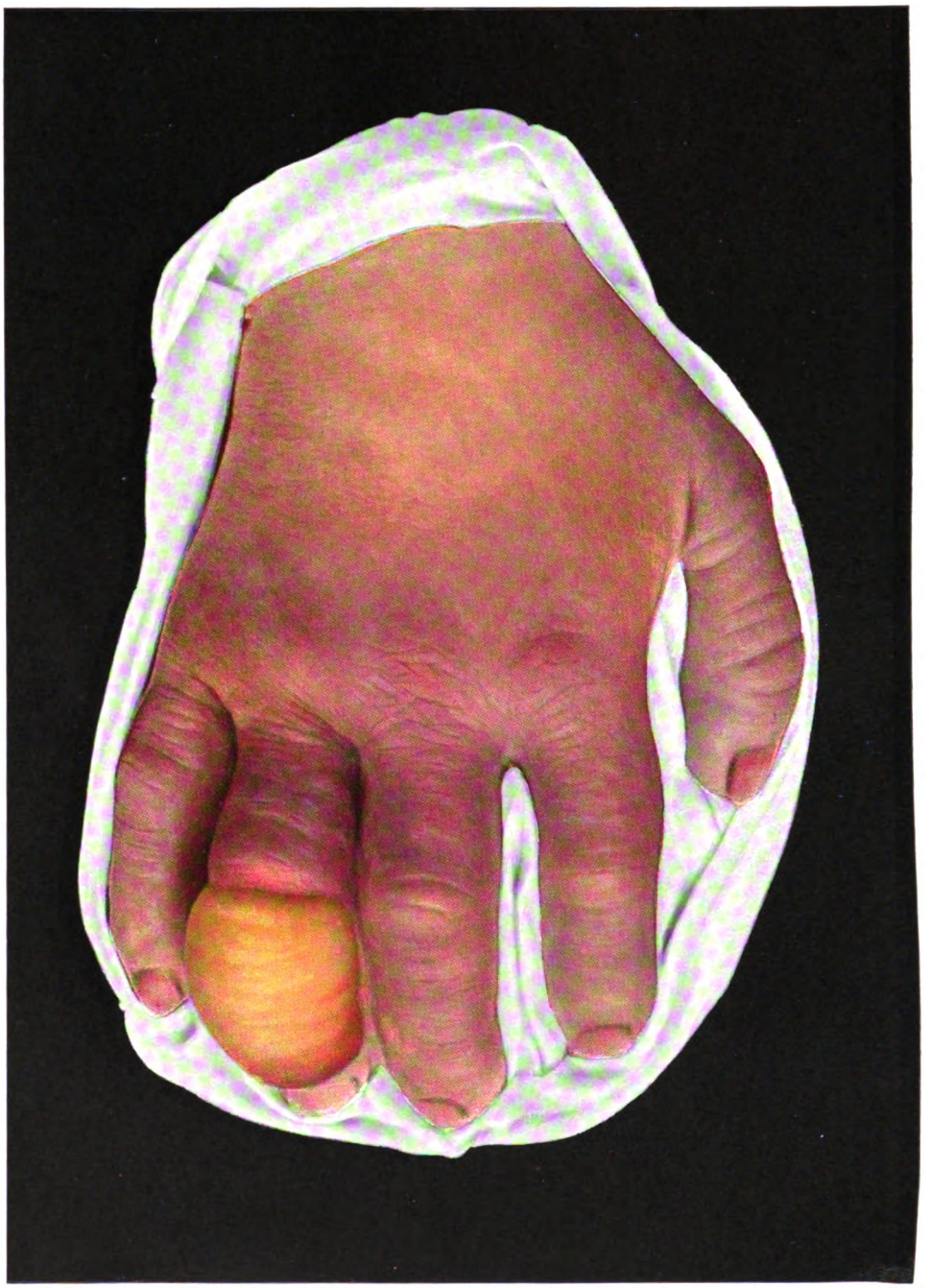

Fig. 137. Congelatio erythematosa — bullosa.

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frostbite of the first degree, however, there is complete recovery.

Longer exposure to cold, or exposure to more severe cold, causes venous congestion, œdema, and the formation of blisters. The skin becomes blue or white, cold and insensitive, and is often covered with numerous blisters, with bluish-black contents; after their rupture the exposed corium is dark in color and very painful. Infection is liable to occur, causing extensive ulceration with little tendency to heal, and leading to cicatricial contraction. Pain is more severe and continuous in frostbite of the second degree.

In frostbite of the third degree, in the same way as in burns of the third degree, there is gangrene of the skin and necrosis of the deeper tissues, due to thrombosis of the vessels. The skin is at first bluish black, cold and insensitive, later on quite black. Separation of the frozen tissues may take place either by dry or moist gangrene. The zone of demarcation has often a putrid character. Progressive phlegmonous inflammation may spread from the borders of the frozen area, and may lead to general infection. Along with frostbite of the third degree the neighboring parts are affected in the first and second degrees, and other parts are ulcerated; so that the clinical picture is variegated. The gangrenous and necrotic parts, after some months, are cast off spontaneously. The nails soon fall off in frostbite of the hand. In frostbite of the third degree, parts which at first showed signs of the second degree only, may afterwards become gangrenous.

Healing eventually takes place by the formation of very unsightly hypertrophic scars, which may cause contractures. Contractures may also be caused by paralysis of nerves, or by waxy degeneration of muscle fibres. Frostbite is said to cause changes in the blood-vessels which may lead to secondary gangrene. The general condition of the patient is little impaired in acute local frostbite of circumscribed regions.

The period of healing varies according to the degree of the frostbite, but is usually longer than in burns and causes more severe after effects.

General frostbite is common in severe winters among weary wayfarers who weaken their power of resistance to cold by alcoholic drinks. After the preliminary feeling of cold they become overcome by fatigue, fall down and become frostbitten. People may even fall unconscious without any previous symptoms. The body, lying on the ground, becomes cooled to below 20° C. (68° F.). Exposed parts may even become frostbitten by slight degrees of cold, acting continuously on the recumbent body. The nose, ears and hands then become frozen to ice and fall off when touched, while the blood becomes decomposed and contains ice crystals. This condition may last for days before death takes place. Only early attempts at resuscitation can do any good in these cases. The heart is, however, so weakened that, even if the patients recover consciousness, they succumb some days later with delirium, coma and heart failure. The prolonged action of intense cold may freeze not only the external parts of the body but may convert all the fluid parts to ice. The expansion caused by the conversion of liquids to ice then ruptures the surface of the body.

Patients who have been exposed to general frostbite must not be suddenly warmed, as this may cause death from shock. The stronger the patient's constitution the better is the chance of recovery; but the prognosis of general frostbite is very unfavorable. Extensive paralysis (hemiplegia and paraplegia) may remain after recovery from the immediate effects, and the patients may suffer for years from headache, pains in the joints, and a tendency to local frostbite due to changes in the arteries. [These secondary phenomena may be due to frostbite acting as an exciting cause on pre-existing latent disease, especially disease of the arteries.

Differential Diagnosis. Frostbite may be mistaken for burns in the absence of any history.

Treatment. Chilblains may be treated by hot air apparatus or hot sandbaths, together with general treatment of chlorosis by iron and arsenic. The irritation may be relieved by painting with tincture of iodine, balsam of Peru, or by inunction with bromocoll ointment. Ulcers are best treated with *Hebra's* diachylon ointment. Recurrence can be limited by prophylactic measures.

In acute local frostbite the parts must be warmed gradually—by rubbing with snow or cold applications. Early treatment in this way may restore the frozen skin. In frostbite of the second degree, large blisters should be opened and broken blisters removed. Ulcers should be treated with strict asepsis, and dressed with sterile gauze or ointment. The extremities should be suspended on splints, avoiding all pressure.

In cases with moist gangrene and putrefactive phlegmonous inflammation, early amputation is often necessary to prevent general infection. In dry gangrene, amputation may be deferred till a zone of demarcation has formed. Plastic operations are often required after spontaneous separation of gangrenous parts of the fingers or toes. Morphia injections may be necessary for the severe pain in the early stages of frostbite. Paralysis may be improved by electricity, and contractures by massage; but the latter more often require a secondary operation.

In general frostbite the body must be very gradually warmed. The patient is placed in a cool room and rubbed down with cold water. He is then put in a tepid bath the temperature of which is gradually raised in the course of several hours. If respiration has stopped, artificial respiration must be performed. Injections of camphor and subcutaneous infusion of salt solution is useful to stimulate the action of the

heart. When the patient recovers consciousness hot alcoholic drinks should be given. Local gangrene resulting from general frostbite is identical with that occurring in severe local frostbite, and requires the same treatment.

Fig. 137 shows a case of frostbite of the first and second degrees in a workman who had had repeated milder attacks in the winter, after exposure of his hands to cold water during his work. The hands were permanently blue, and in the winter painful chilblains developed on the fingers, especially on the extensor surface. He finally developed frostbite of the second degree, which is shown by the whiteness of the ends of the fingers, and other changes in the fourth finger. The skin over the first joint of the fourth finger is blue, and a large blister containing yellow lymph has developed on the extensor surface of the last joint. The patient complained of severe burning pains in the tips of the fingers, especially in the fourth. The blister was opened and the epidermis replaced on the corium, the hand was dressed with ointment and put on a splint. Under this treatment the skin quickly recovered.

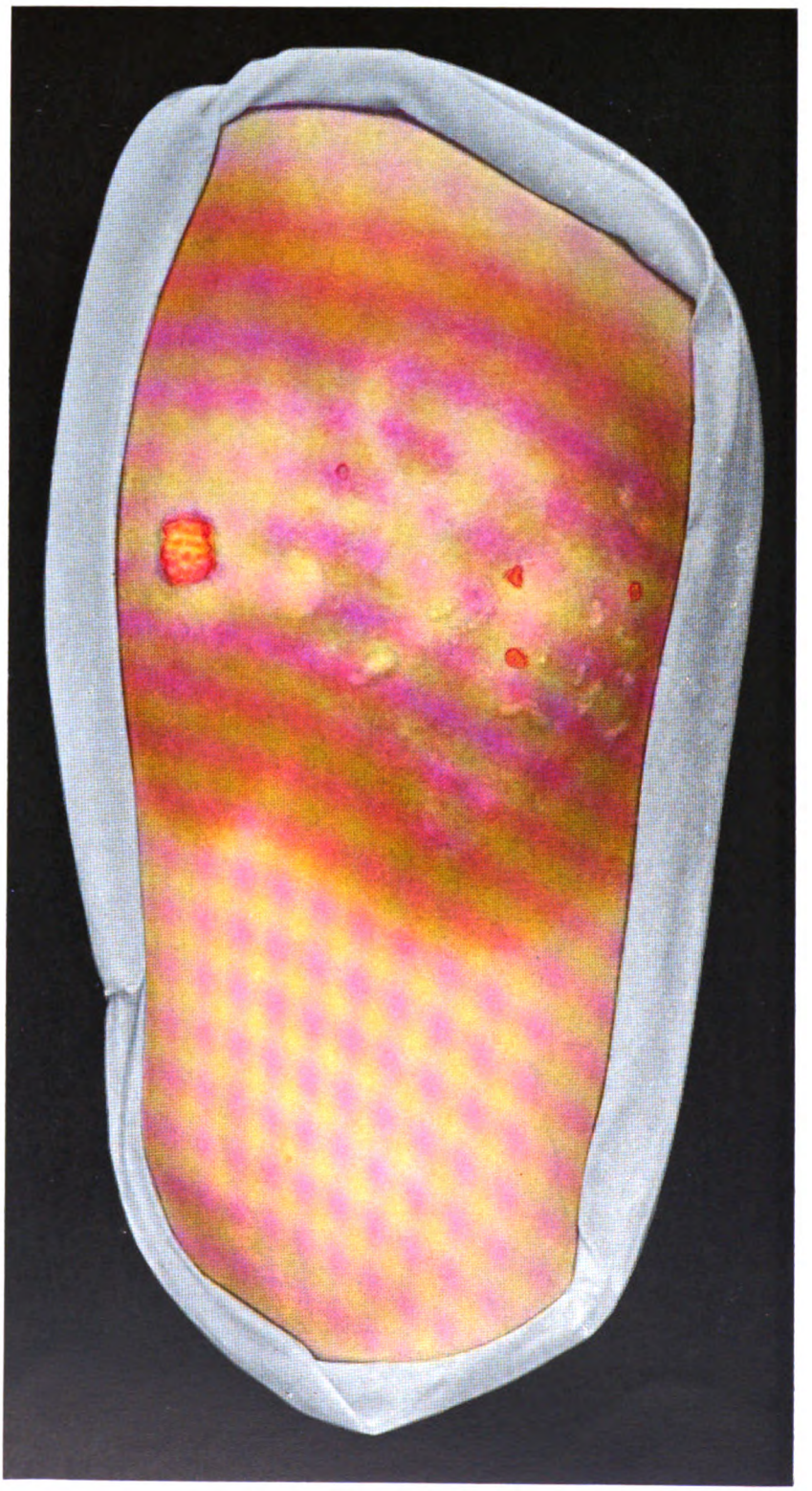

Fig. 138. Combustio (X-Rays).

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X-Ray Burn

Plate CX, Fig. 138.

The X-rays have been used for the treatment of various diseases; sometimes with good results, as in lupus, chronic eczema, etc.; sometimes with no result, as in malignant tumors. A single exposure, properly performed, causes no injury to the skin; but repeated exposures sometimes give rise to changes in the skin, especially when the tubes are placed nearer than thirty centimeters from the skin, and when the exposures are too long or too frequent. The changes produced take the form of a dermatitis, and certain individuals appear to be predisposed to it.

The first signs are redness, swelling and tension of the skin, accompanied by itching and burning sensations. This condition is followed by fissures in the skin and finally ulceration, which is characterized by its chronic and progressive nature. Workers in X-ray laboratories are subject to a chronic form of dermatitis of the hands, unless they protect themselves with gloves, lead-foil, etc. The skin becomes dry, cracked and fissured; the nails become brittle and are often shed. Some cases become gangrenous, and the necrotic tissue is separated by a zone of demarcation. Other cases develop into carcinoma and require amputation of the hand. Some cases are fatal from exhaustion.

X-ray dermatitis can be prevented by placing the tube not less than thirty centimeters from the skin, and by avoiding too long or too frequent exposures.

Workers in X-ray laboratories should take all possible precautions, by the use of lead-foil, gloves, etc. The mode of action of the rays is still not quite clear. Cases have been observed in which exposure to X-ray has caused atrophy of the testicles, interruption of pregnancy, etc.

Treatment. In mild cases, due to the action of a single exposure on a sensitive skin, the action of sea air is said to be beneficial. Chronic X-ray dermatitis is very rebellious to all the usual form of treatment.

Fig. 138 shows an X-ray burn which followed a long exposure made for a swelling of the thigh. The skin became red, then white, and finally ulcerated in several places. The brown coloration indicates healing of the less-affected parts. The ulcers healed after the application of simple dusting powder.

This case is interesting because the X-rays, which were applied to a peripheral sarcoma of the femur, not only caused no improvement but aggravated the tumor. This shows the danger of the treatment of malignant tumors by the X-rays, for, as operative treatment is postponed, more extensive operation becomes necessary later on. In this case X-ray examination showed the presence of sarcomatous masses in the soft parts (by bony spicules) necessitating high amputation through the thigh.

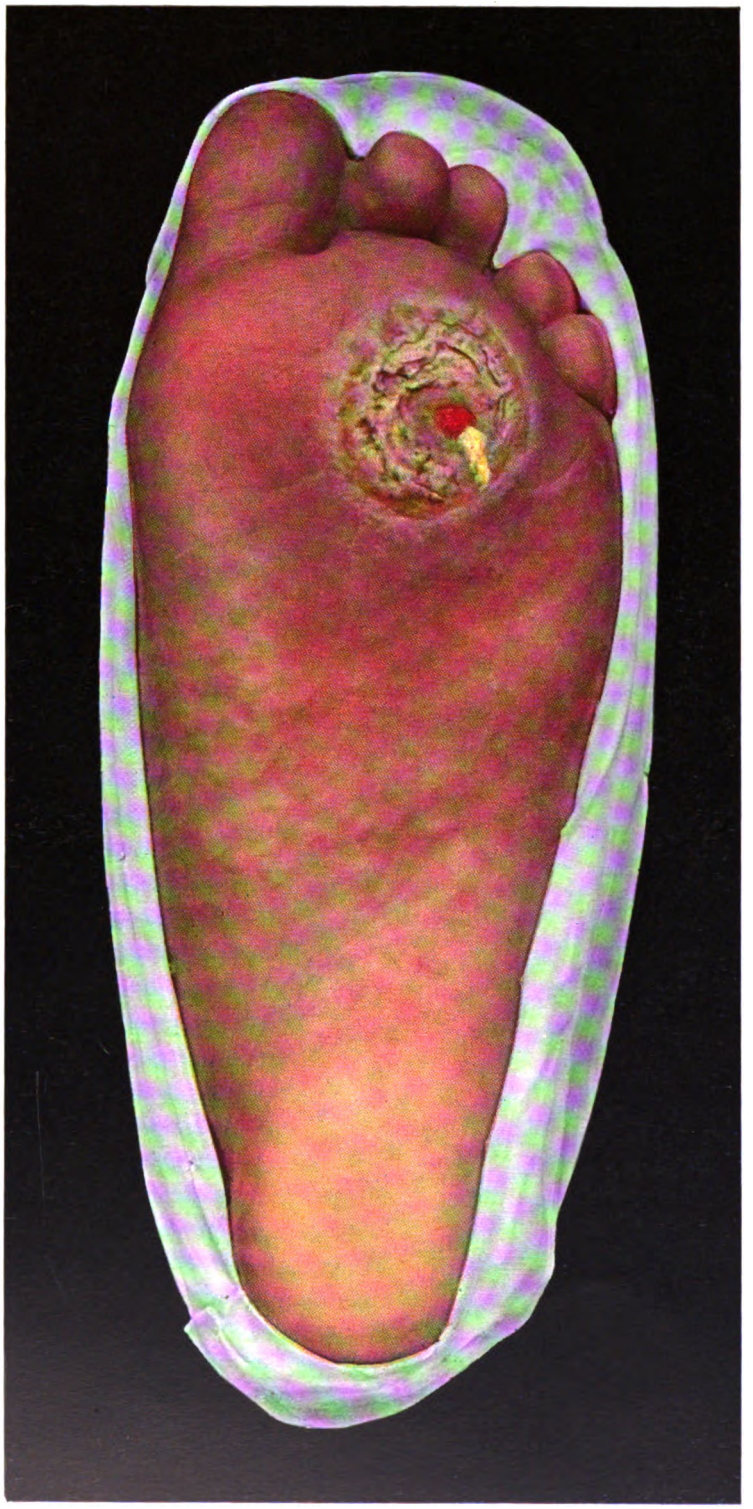

Fig. 139. Mal perforant du pied - Gangraena Raynaud.

Rebman Company, New-York.

MALUM PERFORANS PEDIS (*Perforating Ulcer of the Foot*)
GANGRÆNA RAYNAUD (*Raynaud's gangrene*)
Plate CXI, Fig. 139.

Perforating ulcer of the foot commences as a hard horny thickening of the epidermis over the heads of the third and fifth metatarsal bones, somewhat resembling a clavus but much more extensive. The epidermis becomes fissured and finally ulcerated in the center. The ulcer is characterized by its tendency to extend deeply, and by its persistence in spite of all kinds of treatment. The disease is essentially chronic and leads to destruction of muscles, tendon-sheaths, bones and joints, by continuous crateriform extension of the ulcer into the deeper tissues. The epidermis always remains thickened at the border of the ulcer, and is sometimes undermined. The visible surface of the ulcer is small and is covered with flabby granulation tissue. Necrotic shreds often protrude, indicating extensive necrosis of the fascia and tendons. There is often loss of sensation in the skin for some distance round the ulcer. As a rule there is little pain, but sometimes paræsthesia. The general health may suffer from prolonged suppuration, or the condition may be aggravated by acute progressive phlegmonous inflammation.

Perforating ulcer is of trophoneurotic origin and due to disease of the nervous system. It occurs in tabes, syringomyelia, certain forms of spina bifida (Figs. 143 and 144), and also in diseases where sensation is lost in the lower extremities. Owing to the loss of sensation the patient does not notice the injury to the sole of the foot caused by pressure, and in this way a trophoneurotic ulcer develops, charac-

terized by hard borders due to the horny epidermis which is normally present in the sole of the foot. These ulcers may also develop on the outer border of the foot in cases of paralytic pes varus (Fig. 143). Some authors attribute the condition to disease of the blood-vessels (arteriosclerosis, endarteritis obliterans) as well as to trophoneurotic disorder, and in many cases both conditions are probably present. That the blood-vessels play a part in the pathology of perforating ulcer is supported by the fact that this condition is often met with in syphilitics and alcoholics with vascular disease.

Raynaud's gangrene—which is better called local asphyxia, as it only consists in the first stage of gangrene—is usually symmetrical, and affects the feet more often than the hands. After a short premonitory stage during which the digits become cold and white (vaso-motor constriction), the tips of the fingers or toes become dark-purple and the proximal parts red (vaso-motor paralysis). The disease is due to vaso-motor disturbance depending on disease of the peripheral or central nervous systems. The symptoms consist in paræsthesias and disturbance in the temperature sense, and pain on changes of temperature.

Differential Diagnosis. A commencing perforating ulcer may be mistaken for a clavus complicated by a mucous bursa and central fistula; but the latter does not extend so deeply.

Raynaud's disease may be confounded with the early stages of other forms of gangrene (Figs. 132, 133 and 140), or frostbite; but the changes in Raynaud's disease are diffuse and symmetrical.

Treatment. Even in the early stage of perforating ulcer, removal of the callosity and necrosed tissue gives little result. In the later stages no treatment is of any use. The wound must be protected from

infection by aseptic dressings. In some cases partial amputation of the foot is necessary, especially when there is extensive necrosis of the plantar fascia (*Lisfranc's*, *Chopart's* or *Pirogoff's* amputations). If there is phlegmonous inflammation free incisions must be made down to the bone. Amputation through the leg may be necessary in cases of progressive phlegmon or general infection. Internally iodide of potassium should be administered, and other treatment for arteriosclerosis (Fig. 140).

In *Raynaud's* disease exposure to cold must be avoided. Treatment by hot air, hot sand-baths and massage is useful.

Fig. 139 shows a case of perforating ulcer of the foot in a typical position, over the head of the third metatarsal bone. The epidermis is fissured and thickened round the small ulcer, which is covered with granulations. A piece of necrosed fascia is seen protruding from the ulcer. The peripheral part of the foot shows diffuse bluish-red coloration, which was also present symmetrically on the other foot (*Raynaud's* disease). The ulcer showed no tendency to heal under treatment by aseptic dressings and rest in bed, so amputation was performed at the tarso-metatarsal joint.

GANGRÆNA DIABETICA (*Diabetic gangrene*) ARTERIOSCLEROSIS

Plate CXII, Fig. 140.

Diabetes mellitus greatly diminishes the power of resistance of the body against infection. Various pyogenic affections, such as furuncle, carbuncle, abscesses (*e.g.* mammary abscess, Plate V) or extensive phlegmons may develop in diabetic patients after comparatively slight causes, especially in the lower extremities. The dry, irritable skin of diabetics is liable to infection through scratches. Moreover, the sugar-containing tissues are favorable to the growth of bacteria, which are thus able to cause progressive phlegmonous inflammation. Putrefactive phlegmon is more common than pyogenic phlegmon in diabetics, and gives rise to moist gangrene of the skin, necrosis of the deeper tissues, and often general infection. Dry gangrene may also develop suddenly in the lower extremities in diabetics affected with arteriosclerosis. In this way, the whole leg may be affected with dry gangrene from thrombosis of the popliteal artery. The first symptoms are pain, numbness and tingling sensations in the toes. One or more toes then become bluish black and cold, later on bluish gray (Fig. 140); while the skin on the dorsum of the foot is red and œdematous. In this stage there are often severe neuralgic pains, while the general condition of the patient is impaired by increase of sugar in the urine, sleeplessness, headache and exhaustion. In old diabetics with dry gangrene of the toes demarcation may take several months to develop. Dry gangrene may always change to moist, the latter progressing more rapidly.

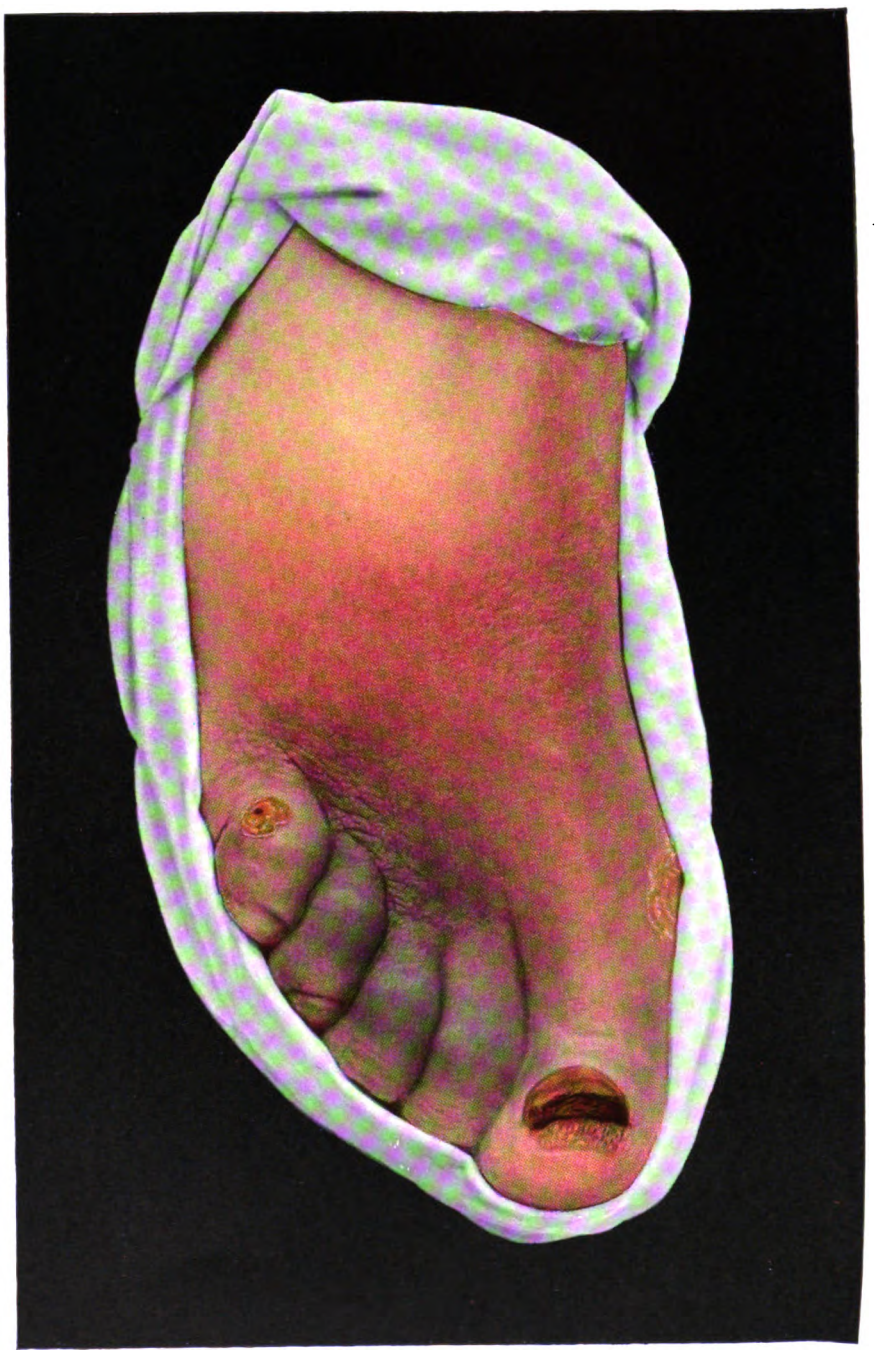

Fig. 140. Gangraena diabetica — Arteriosklerosis.

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The prognosis in these cases is bad, especially when there is much sugar in the urine. Death may occur from heart failure, general infection or diabetic coma. When the general treatment of diabetes fails to act, the gangrene usually extends, and leads to death.

Prophylaxis consists in the early diagnosis and treatment of diabetes. It is, therefore, important to examine the urine for sugar in all cases of pyogenic and putrefactive infections. Diabetic patients should pay strict attention to bodily cleanliness and try to avoid all kinds of infection. They should also avoid the causes which lead to arteriosclerosis.

Differential Diagnosis. Diabetic gangrene is distinguished from other forms of gangrene by examination of the urine. Extensive calcification of the arteries can sometimes be seen by X-ray examination.

Treatment. In dry gangrene it is best to wait for demarcation, unless extensive arteriosclerosis is present. If, however, the popliteal artery is pulseless, amputation of the leg is the only remedy. If there is no arteriosclerosis the gangrene may slowly extend for months. When demarcation is complete amputation may be performed directly above the line of demarcation. Before demarcation the parts should be treated with dry aseptic dressings (moist dressings cause putrefaction), and be suspended. In slowly extending moist gangrene demarcation may be waited for if the temperature does not remain high. In rapidly extending moist gangrene with high temperature early amputation is indicated some distance above the gangrene. In gangrene of the lower extremity with arteriosclerosis it is better to amputate through the thigh; for the flaps after amputation through the leg are badly nourished even in healthy individuals, and in diabetics they are liable to become gangrenous. Amputation through the thigh is best performed above

the condyles (supracondylar amputation), or through the epiphyseal line. Epiphyseal stumps have considerable supporting power. As a rule, amputation may be conservative in slowly progressing cases which are not complicated by phlegmonous inflammation, arteriosclerosis or high temperature. On the other hand, rapidly extending gangrene complicated by arteriosclerosis and phlegmon always requires high amputation.

It is best to give an injection of scopomorphine (*Riedel's* preparation) before the operation; less quantities of chloroform or ether are then required. In these cases both general anæsthesia and lumbar anæsthesia are badly borne, and infiltration anæsthesia is contra-indicated, as it causes inflammation of the weakened tissues. When the vessels are affected with arteriosclerosis they should be compressed by the fingers of assistants during the operation, as the application of the elastic tourniquet may cause thrombosis. The wound should be dressed with sterile gauze; iodoform is contra-indicated on account of the danger of iodoform poisoning. Primary suture of the flaps should not be attempted, and these should, therefore, be made larger than usual. Secondary suture of the flaps may be performed after a few days if the progress of the case is satisfactory. Ligatures must not be applied too tightly to vessels affected with arteriosclerosis, as the coats of the vessel may give way and cause secondary hemorrhage. The operation must be performed under the strictest aseptic precautions, as the diabetic tissues are easily infected, and osteomyelitis may occur in the bone stump or phlegmonous inflammation in the soft parts.

After the wound has healed ulceration is common in the amputation stump. This must be treated with aseptic dressings to avoid fresh phlegmonous inflammation. In some cases amputation of both legs may be necessary for gangrene of both the feet. Only about fifty per cent. of cases of diabetic gangrene

recover after amputation, a great many cases succumbing to diabetic coma. Whenever possible the amount of sugar should, therefore, be reduced by general treatment of the diabetes before operation. Cases where acetone is present, and which give a positive result with the perchloride of iron reaction, have an unfavorable prognosis. The general condition requires treatment by strict diet and the administration of salicylate or bicarbonate of soda in large doses. Subcutaneous injection of saline solution may be tried in diabetic coma. Thirst may be relieved by tincture of opium or by *von Bergmann's* diabetic drink (citric acid 10; glycerin, 100; distilled water, 1,000).

ARTERIOSCLEROSIS (*Atheroma*)

This disease consists in the thickening of the walls of the vessels by connective tissue formation, with subsequent fatty degeneration of the inner and middle coats (atheroma) and the deposition of calcareous plates, causing roughening of the inner surface of the vessel and leading to thrombosis. The disease is more common in the male sex. Central and peripheral nervous affections, especially those causing vaso-motor disturbances; infective diseases, including typhoid, malaria, syphilis, general infection, leprosy, gout and diabetes; the action of alcohol, nicotin and lead; overexertion and sudden exposure to cold have all been cited as causes of arteriosclerosis. [The term *arteriosclerosis* is here used to describe what is generally known in England as *atheroma*. The fundamental cause of this is generally considered to be syphilis, though other causes mentioned above probably contribute. General arteriosclerosis, characterized by a general fibroid thickening of all the arteries, is of more complex etiology, the chief factors being probably syphilis, chronic alcoholism, infective fevers, gout, and microbial toxæmias].

A tortuous condition of the temporal and radial arteries is often present in arteriosclerosis, along with differences in the pulse in different arteries. Extensive calcification is sometimes visible by X-ray examination.

The symptoms begin with pains of a rheumatic character. The feet, in which the disease often begins, are blue, cold and dry. Sensations of numbness and tingling are often present. There may be severe pain in the heels, preventing the patient from

walking (*Charcot's* intermittent claudication). Extensive arteriosclerosis may cause gangrene of the lower extremities. In women arteriosclerosis more often affects the hands causing great pain and loss of function; but gangrene in the hands is very rare. Arteriosclerosis of the cerebral arteries causes severe headaches, attacks of loss of consciousness, or cerebral hemorrhage.

Differential Diagnosis. Commencing arteriosclerosis of the extremities with no visible change in the vessels may be mistaken for gout or rheumatism, etc. In advanced cases the diagnosis is easy, owing to the hardness of the vessels.

Treatment. Prophylactic treatment consists in avoiding, as far as possible, the causes which may lead to arteriosclerosis. The best therapeutic measures are those which promote metabolism and strengthen the heart; for instance, light gymnastics, massage, mud baths, sand baths, Wiesbaden hot springs, etc. Internally iodide of potassium should be administered. Hot air treatment and hot potash baths are useful for the pains in the heel. In severe cases morphia may be necessary.

Fig. 140 shows commencing gangrene of the right foot in a man of fifty-six, suffering from diabetes for some years. The toes are bluish red in some parts, grayish black in others, while the dorsum of the foot is red. The skin was pale and cold. The discoloration appeared in the course of a few hours, and in a few days extended to the ankle joint. Moist gangrene spread rapidly from the toes, and lymphangitis extended up the leg.

The X-rays showed numerous calcareous deposits in the anterior and posterior tibial arteries. Amputation was performed above the knee joint, after the sugar had been reduced from five to two per cent. by

three days' treatment of the diabetes. After operation the sugar diminished still further, and the temperature fell—two favorable signs. Secondary suture of the stump was performed on the fifth day and the wound healed in four weeks. After general treatment of the diabetes the sugar disappeared from the urine.

The figure also shows other changes. On the inner side of the foot over the metatarsophalangeal joint is a large clavus, and another on the fifth toe. The nail of the great toe is affected with onychogryposis, a common condition in old people who neglect their feet. As the nail caused trouble in walking, it was removed under local anæsthesia.

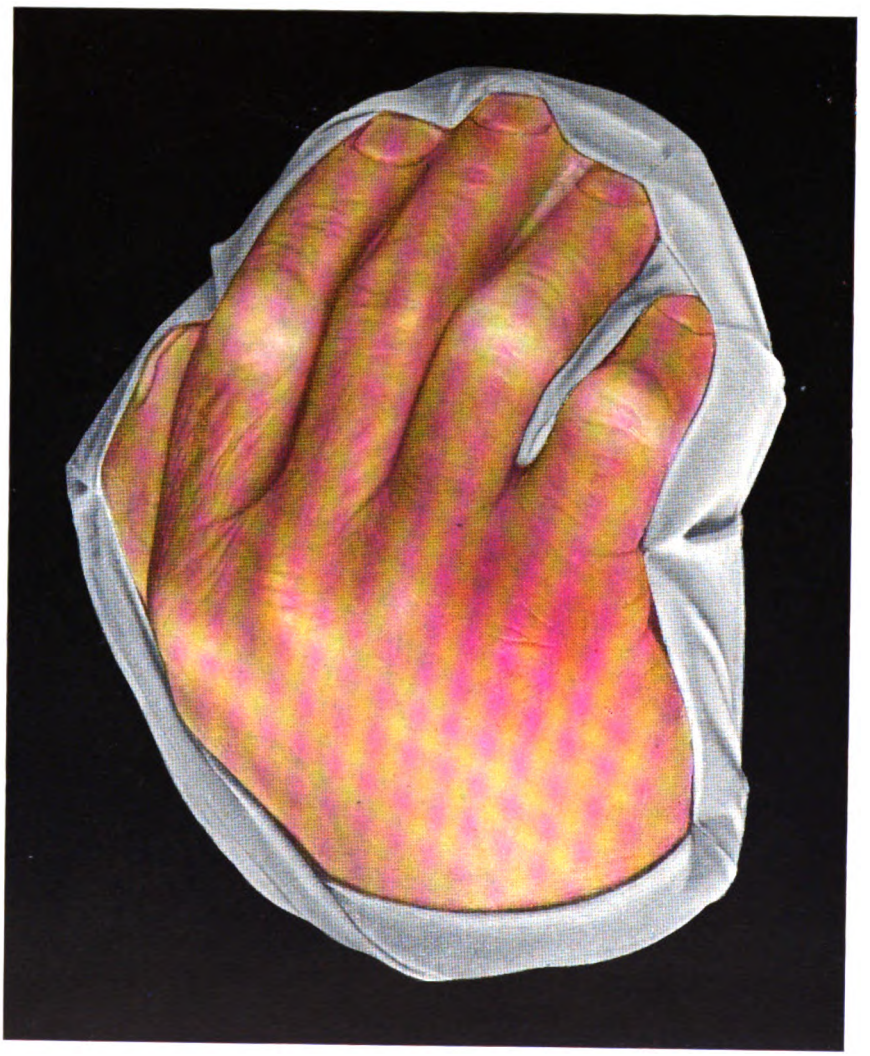

Fig. 141. Arthritis urica.

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ARTHRITIS URICA (*Gouty Arthritis*)

Plate CXIII, Fig. 141.

Gout is a disorder of metabolism which is often transmitted from father to son for generations. It therefore usually occurs among people with a hereditary predisposition. It most often affects middle-aged men who indulge in high living and who take too little exercise.

The disease is due to the deposit of urate of soda in various places, especially in the cartilages of the joints. According to *Pfeiffer* there is no increase in the formation of urate of soda, but only deficient elimination. The urate of soda deposits form yellowish-white masses in the cartilage, synovial membrane, tendons, subcutaneous and periarticular tissue, bursæ, bronchi, intestinal mucous membrane and kidneys—in fact, in all the tissues and organs of the body. An acute attack of gout is caused by deposit of urate of soda in a joint, usually the metatarso-phalangeal joint of the great toe (*Podagra*). The symptoms are great pain in the affected joint, slight rise of temperature and a certain amount of constitutional disturbance (gastric pain, nervous phenomena, rheumatic pains, etc.). The first attack is sometimes excited by an injury to the foot. The region of the joint is swollen and œdematous, and the skin shows erysipelatous reddening and phlegmonous infiltration. The slightest touch or movement causes intense pain. There is slight effusion in the joint. After some hours the pain subsides, but generally recurs on the second night; and so on for about two weeks, till the attacks gradually become less painful and finally disappear. Slight swelling of

the affected joint remains. Later on fresh attacks may occur, often after many years. During the attacks there is always a heavy sediment in the urine. Repeated attacks may give rise to a permanent nodular swelling of the joint, and slight trauma may bring on another acute attack (*e.g.* hand pressure on gouty fingers).

Chronic gout, which is rarely primary and generally results from the acute form, is observed also among the poorer classes. It often affects the joints, but is less painful. The frequency with which the metatarso-phalangeal joint is attacked is perhaps due to bad circulation of the blood, owing to its peripheral position. This joint is also affected by arthritis deformans in old people. Large deposits of urate of soda give rise to gouty nodules or tophi, which occur in the joints of the fingers, hand, foot and elbow. They also occur in the cartilages of the ear, nose and eyelids in the form of small, yellowish nodules, which become hard and painful. In advanced cases of gout these nodules may be found in all the joints and cartilages, joint capsules, tendon-sheaths, cartilages of the ribs, and in other tissues.

Microscopic examination of gouty deposits shows the presence of crystals of urate of soda. These crystals act on the tissues like foreign bodies, and cause not only pain but gradual necrosis by pressure. The necrosed tissues are expelled by the formation of fistulas, and through the latter infection of the joints may take place. Joint infection may also occur by way of the blood (staphylococcal or streptococcal infection), without communication with the exterior. Suppuration in a gouty joint is always serious, as it easily leads to general infection. The cartilages of the joint may be destroyed by the gouty deposits, without the occurrence of suppuration, and lead to subluxation and anchylosis. Tophi, especially when situated in the subcutaneous tissue, may give rise to ulceration, venous thrombosis and phlebitis, espe-

cially in the lower extremities. Eczema of the skin is common in gouty subjects.

Although in most cases of gout the joints are affected, and the symptoms are those of joint inflammation, gouty deposits in other tissues and organs may give rise to the most diverse symptoms. Deposits in the tendo Achillis causes achylodynia with pain in the heel; deposits in other places may cause sciatica and lumbago, asthma and bronchitis, iritis and other affections of the eye, disorders of the intestine, etc.

In all long-standing cases of gout there is a danger of complications affecting the internal organs. The chief of these is chronic interstitial nephritis, in which numerous deposits of urate of soda are found in the kidneys, which may give rise to renal calculus. Gouty subjects are also liable to emphysema of the lungs. The prognosis in cases of pronounced gout is always doubtful.

Differential Diagnosis. Gouty arthritis is most often confounded with chronic rheumatism, but in the latter the skin over the joints is unchanged. In purulent arthritis there is high temperature and rigors while the temperature in gout does not exceed 38° C. (100° F.) provided no suppuration is present. Enchondromas of the fingers (Fig. 50) differ from gouty deposits by the absence of pain. Gout of other organs must be diagnosed by the history of the case. Large deposits of urate of soda can be seen by X-ray examination; *e.g.* in bursæ.

Treatment. Persons who are predisposed to gout should try to avoid it by careful living, exercise, etc. In acute gout, tincture of colchicum should be given in large doses (fifty to one hundred drops daily). The affected joint should be wrapped in wool and suspended on a splint. Hot air treatment is also useful. If suppuration occurs in the joint (with high

temperature and rigors) arthrotomy must be performed under strict aseptic precautions. In some cases resection of the joint may be necessary. General infection is common in such cases.

During the acute attack the patient should avoid meat, eggs and alcohol, and drink plenty of alkaline waters. Purgatives are also indicated.

Ice bags and moist fomentations should be avoided, as the former may cause necrosis of the skin and the latter maceration. Massage is contra-indicated. Internally, ten to twenty drops of hydrochloric acid may be given daily; salicylate of soda, aspirin and iodide of potassium are also useful. Phenacetin may be given for the pains, or morphia in severe cases.

When there are frequent attacks of gout treatment at the various springs is useful (Wiesbaden, Karlsbad, etc.). The diet should be carefully regulated—plenty of vegetables, especially celery; little carbohydrates, little meat, little alcohol and no beer.

Fig. 104 shows a case of acute gouty arthritis affecting the metacarpo-phalangeal joint of the second finger. The whole joint is swollen and very painful to touch and on movement. Tophi are present on the other metacarpo-phalangeal joints and on the interphalangeal joints of the second to the fifth fingers. The skin over the tophi is white from pressure. The patient, whose grandfather was gouty, had suffered for years from gouty arthritis in the joints of both hands.

Malformations

ENCEPHALOCELE OCCIPITALIS (*Occipital Encephalocele*)
RHACHISCHISIS
Plate CXIV, Fig. 142.

MYELOCELE—PES VARUS
Plate CXV, Fig. 143.

MYELOCYSTOCELE—MYXOLIPOMA
Plate CXVI, Fig. 144.

LYMPHANGIOMA (*Congenital multiple*)
Plate CXVI, Fig. 145.

TERATOMA MONOGERMINALE (*Monogerminal Teratoma*)
Plate CXVII, Fig. 146.

DUCTUS OMPHALO-MESENTERICUS PERSISTENS
(*Persistent omphalo-mesenteric duct*)
Plate CXVIII, Fig. 147.

HERNIA FUNICULI UMBILICALIS CONGENITA
(*Congenital Umbilical Hernia*)
Plate CXVIII, Fig. 148.

AMPUTATIONES AMNIOTICÆ (*Amniotic Amputations*)
Plate CXIX, Fig. 149.

AKROMEGALIA (*Acromegaly*)
MAKROMELIA
MAKROGLOSSIA
Plate CXX, Fig. 150.

The study of malformations (teratology) is of great interest to the surgeon, because many of these can be improved by surgical intervention. A knowledge of embryology is necessary in order to understand malformations. We distinguish between primary malformations which affect the embryo in its early stages of development, and secondary malformations which affect a part already formed, by some influence acting on it during intra-uterine life. The latter are spoken of as arrested development. Slight disturbances in development are called anomalies; greater deformities, malformations. The greater the mal-

formation, the earlier was its origin. The causes which lead to malformation may be already present in the embryo, or arrested development may be due to external causes. Experimental observations on animals have shown that malformations may be caused by injury. In the lower extremities malformations may be caused by pressure or by abnormal positions of the fetus in the uterus (various forms of talipes—pes varus, pes valgus, pes calcaneus). Pressure on the fetus may be caused by a uterine tumor or by deficiency in the liquor amnii, and signs of such pressure can often be seen after birth of the child. Many malformations are due to anomalies in the membranes; *e.g.* amniotic adhesions. All malformations caused in this way are cases of arrested development. These amniotic adhesions or bands may prevent the union of parts which should normally become united (branchial clefts) or may cause duplication of parts, or partial or ccmplete separation (amniotic amputations, aberrant glands).

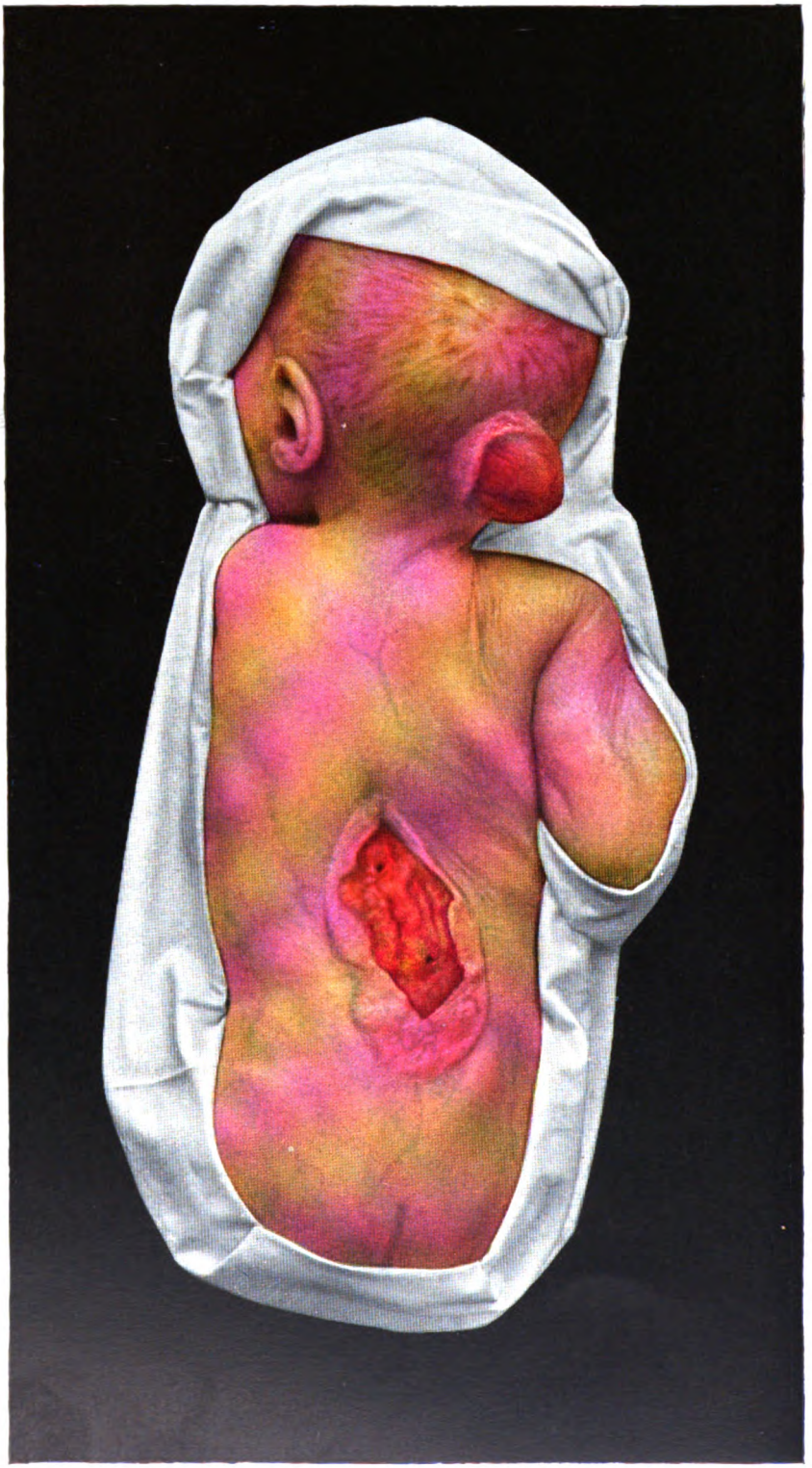

Fig. 142. Encephalocele occipitalis – Rachischisis.

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ENCEPHALOCELE OCCIPITALIS (*Occipital Encephalocele*) RHACHISCHISIS

Plate CXIV. Fig. 142.

Encephalocele, or cephalocele, is a malformation due to arrested development, and occurs in two regions—the region of the nose (syncipital encephalocele) and the occipital region (occipital encephalocele). The former is subdivided into naso-ethmoidal, naso-frontal and naso-orbital; the latter into superior and inferior occipital encephalocele, according as it is situated above or below the occipital protuberance. According to *Müller*, one case of encephalocele occurs in thirty-six hundred births. The deformity is due to more or less extensive deficiency in the closure of the cerebro-spinal canal, caused by trauma or by amniotic bands. The earlier this occurs in fetal life the more extensive is the cleft in the cerebro-spinal canal. In extensive cases there may be acrania or anencephalus, while in slighter degrees there is only a defect in the bone and dura mater. Owing to the defect in the dura mater there may be prolapse of the brain through the bone, generally a hernial protrusion of one of the ventricles. According to *von Bergmann* the existence of a true congenital meningocele in which the dura is intact, and there is only a hernial protrusion of the membranes through the gap in the bone, must be regarded as doubtful. The author's observations on myelocele (Fig. 134) have also shown that the inner covering of the protrusion, which is said to be dura, often consists of connective tissue only, and that the inner wall is often formed of ciliated columnar epithelium, and, therefore, represents the degenerated ventricle

of the brain. Hence the so-called meningocele is a true encephalocele or myelocystocele (Fig. 144).

As the subdivision of the different forms into meningoceles, encephaloceles, encephalomeningoceles encephalocystoceles and encephalocysto-meningoceles depends on pathological anatomy, and cannot be distinguished clinically, it is sufficient for all practical purposes to use the term encephalocele or cephalocele for all hernial protrusions through the skull, especially as they mostly contain a protrusion of the ventricle. For instance, the so-called encephalomeningocele has been shown to be not a true meningocele, but a cystic formation which has become gradually cut off from a primary hernia cerebri or encephalocele.

Cephaloceles occurring at the sagittal suture, the fontanelles or other parts of the skull are, according to *von Bergmann*, either dermoids or caused by trauma after birth (spurious traumatic acquired cephalocele). Congenital cephaloceles are either syncipital or occipital. Syncipital cephaloceles have generally a wide base, while occipital cephaloceles are pedunculated. Occipital cephaloceles may attain a large size—as large as the child's head. The skin at the base of the tumor is thickened and covered with radially arranged hair. The tumor may be covered with normal skin, but more commonly most of the surface resembles fresh scar tissue; or, when ulceration is present, it resembles the mucous membrane of the intestine. Vascular anomalies—telangiectases and angiomas—are often present. The tumor is diminished by pressure, and can be completely emptied in cases when it apparently consisted of a collection of fluid only. After the tumor has been emptied by pressure the hole in the skull can be felt, situated symmetrically in the middle line. It is generally small and circular, and can sometimes be shown by X-ray examination. As the tumor can be diminished by external pressure, so is it increased

by internal pressure; *e.g.* when the child cries. Cystic cephaloceles may be translucent. In other cases there is little diminution on pressure. Irregular partitions can then be felt in the interior of the sac. Firm pressure then usually causes bulging of the fontanelle, or sometimes convulsions. Sometimes pulsation is observed in cephaloceles. The skull in these cases is generally very small, and often flattened. Other malformations are often present. The infants are weakly and have a subnormal temperature. The prognosis is generally unfavorable, but is better in cases where the cephalocele can be completely emptied of fluid by pressure, and when no brain substance can be felt in the sac after evacuation of the fluid. Cases of occipital cephalocele with a large gap in the bone, often extending to the vertebræ of the neck, and protrusion of both occipital lobes and the whole of the cerebellum, are soon fatal.

Differential Diagnosis. Syncipital cephalocele may be mistaken for dermoid or lipoma. Diagnosis depends on the presence of a gap in the bone, diminution of the tumor on pressure and the presence of other deformities. Occipital cephalocele may be mistaken for cephalhematoma, which sometimes occurs on the occipital bone, especially as cephalhematoma may be surrounded by a hard ring at its base caused by the raised periosteum. Cephalhematoma is not diminished by pressure. However, diminution by pressure may be absent in cephalocele if the gap in the bone is occluded. In doubtful cases an operation will settle the diagnosis.

Treatment. Puncture and injection is useless and dangerous in cephalocele. The only rational treatment is a radical operation. The sac is exposed by incision through the skin, separated down to the bone, ligatured and removed. The defect in the bone may be covered in by suturing the periosteum over

it, by a pedunculated bone flap, or by a celluloid plate. In cases where brain substance is present in the sac, the operation can only be performed when the brain substance can be reduced through the gap in the bone without producing symptoms of cerebral compression. Removal of portions of brain still possessed of function may cause dangerous symptoms, but a functionless dropsical protrusion may be removed without danger. Cases of large defect in the skull, with defect in the cervical vertebræ, or cases combined with other extensive malformations, are inoperable. The after-treatment is complicated by the escape of cerebro-spinal fluid, which is always abundant, even after the most careful closure of the bone defect. The dressings therefore require changing several times daily to prevent infection of the wound.

Fig. 142 shows a cephalocele situated symmetrically in the middle line under the occipital protuberance. The skin at the base of the tumor was thickened; over the greater part of the surface it resembled fresh scar tissue, and presented numerous fine ramifying vessels. The tumor could be completely emptied of its fluid contents by pressure, without causing symptoms of cerebral pressure. After this a circular hole in the bone could be felt about one-half centimeter in diameter. This cephalocele could have been completely removed by radical operation, but for the presence of another malformation of the spine which made the condition of the infant hopeless.

In the dorso-lumbar region from the twelfth dorsal to the third lumbar vertebra is a condition known as rhachischisis (spina bifida). This is a condition of arrested development of the spine in which there is absence of closure of the embryonic medullary canal affecting the bones, soft parts, spinal cord and membranes. This malformation may extend the whole length of the spine, and is then known as total pos-

terior rhachischisis; or it may be limited to one portion only.

Rhachischisis represents the most extreme degree of spina bifida (Figs. 143 and 144). It is most common in the lumbo-sacral region, because the medullary groove closes last in this region to form the neural canal. Rhachischisis is usually associated with other extensive malformations such as anencephalus, acrania, absence of vertebral bodies, etc. Three typical zones can be distinguished situated symmetrically on each side of the vertebral column: (1) a circular, peripheral zone of thickened skin, often covered with abundant hair; (2) a middle zone which resembles fresh cutaneous scar tissue, or the serous coat of the intestine, and has hence been called the epithelio-serous zone; a central zone of flabby granulations with a depression at the upper and lower ends, which represents the open and exposed spinal cord. The depressions at each end of the central zone lead to the central canal of the spinal cord. In cases where the spinal cord is much exposed, death soon occurs from meningitis.

Spina Bifida

MYELOCELE—PEDES VARI
Plate CXV, Fig. 143.
MYELOCYSTOCELE—MYXOLIPOMA
Plate CXVI, Fig. 144.

As already mentioned, rhachischisis represents the most extreme degree of spina bifida. If the arrest of development is limited to one, two or three vertebral arches, the cleft spinal cord is not exposed in the vertebral groove as in rhachischisis, but projects in the form of a tumor through the small cleft in the vertebræ, owing to pressure of fluid on its ventral surface. It thus forms a symmetrical tumor in the middle line, with the same three characteristic zones as in rhachischitis, and is known as a *myelocele* (Fig. 143).

There are four kinds of spina bifida, differing in degree according to the date of their appearance in embryonic life. The first and most extensive form is *rhachischisis*, which has already been mentioned. The second form (*myelocele*) appears later and is limited to a smaller extent of the spine, although it may include the soft parts, bones and spinal cord; this forms a tumor-like swelling. The third form (*myelocystocele*) occurs still later in embryonic life, at a time when the spinal cord and the skin have already closed on the dorsal surface of the embryo, but the dura mater and bone have not yet united. The fourth form (*meningocele*) only occurs in the lumbo sacral region where the spinal cord has become the *filum terminale*. Spina bifida occulta, which also

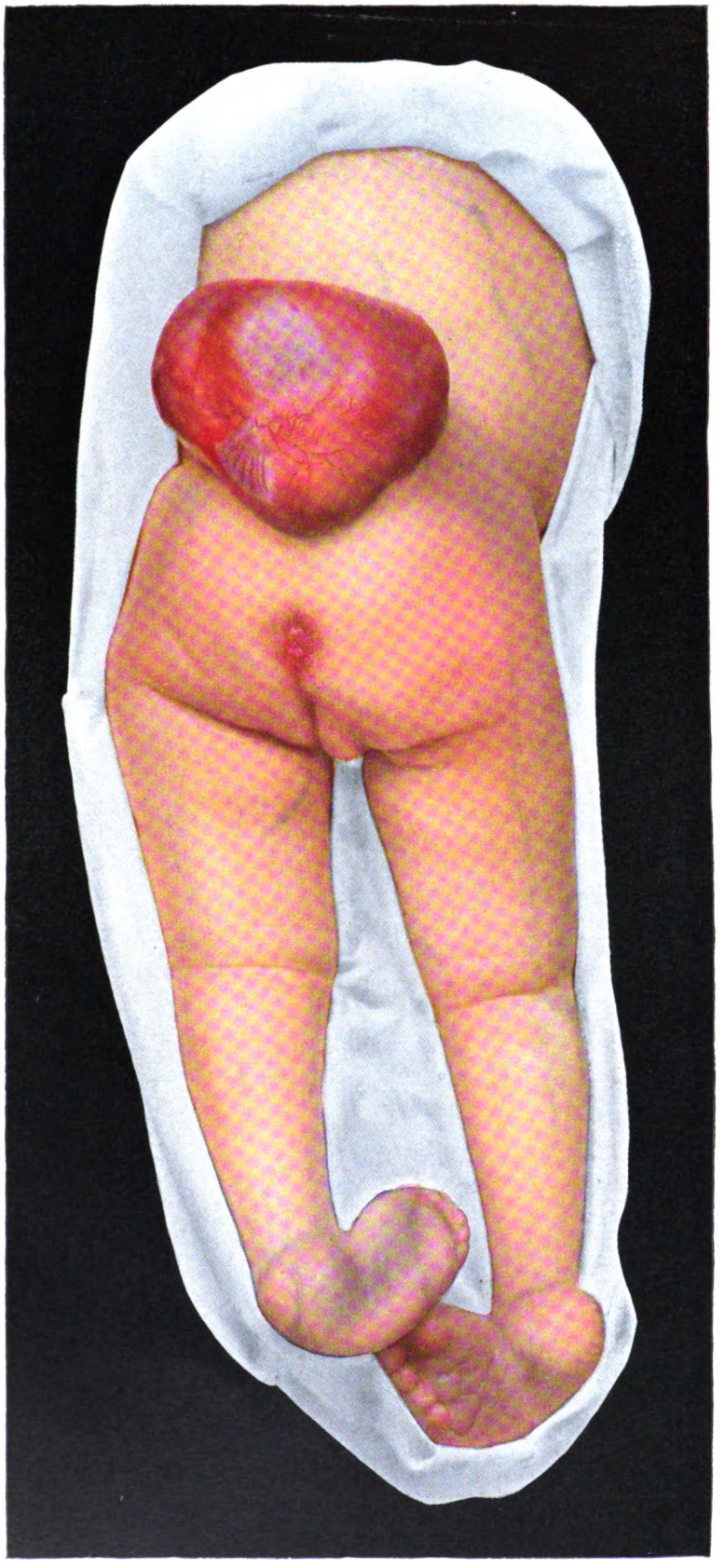

Fig. 143. Myelocele — Pedes vari.

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occurs at the lower extremity of the vertebral column, is not to be regarded as a special form, but as a meningocele.

The subdivision of spina bifida into the three chief forms—myelocele, myelocystocele and meningocele—is the most suitable for practical purposes. Spina bifida is a comparatively rare malformation, occurring in one or one and five-tenths out of one thousand infants.

1. *Myelocele.* By far the most common form is myelocele. According to *von Recklinghausen* this occurs before the twelfth day of embryonic life, as after this time the medullary groove closes to form the neural canal. The arrest of development concerns the dorsal part of the spinal cord and membranes, the vertebral arches, the muscles and the skin. A tumor-like swelling is then formed by the formation of hydrops on the ventral side of the spinal cord which continually pushes the cord out of the vertebral canal through the preformed cleft. According to *von Bergmann* the occurrence of hydrops is due to the absence of dura mater.

Myelocele forms a characteristic swelling with a wide base, situated symmetrically in the middle line, with the three zones already mentioned in the case of rhachischisis; *viz.* an outer zone of thickened skin with abundant hair, and often telangiectases; a second zone of a pink color resembling new scar tissue, with a deep network of ramifying vessels; a third zone of an oval form at the summit of the swelling, red and tumid like intestinal mucous membrane, very vascular, and covered with pus a few days after birth. This third or central zone represents the remains of the cleft spinal cord, and is called the vasculo-medullary zone in distinction to the epithelio-serous or second zone. At the upper and lower ends of the third zone is a depression through which a probe can be passed into the central canal of the spinal cord. These cases generally die from

meningitis through infection of the vasculo-medullary zone. Operative treatment is useless. The spinal nerves become dragged upon by the formation of the protruding myelocele, causing motor paralysis of the lower extremities, bladder and rectum (paralysis of the upper extremities when the myelocele is situated in the upper part of the spine).

The common occurrence of pes varus in these cases (Fig. 143) is due to the myelocele being usually situated at the junction of the lumbar vertebræ with the sacrum where the nerves arise which supply the anterior and posterior tibial muscles; *viz.* the fourth and fifth lumbar and the first and second sacral nerves. Sensory disorders are rare in myelocele, but trophoneurotic disorders occur in the form of extensive eczema and decubital ulcers, especially on the feet; in pes varus on the outer border of the foot.

Diagnosis is easily made by the characteristic appearance, the presence of fluctuation and the cleft in the bone. There is no diminution in the swelling by pressure owing to the absence of communication with the subarachnoid space. Myelocele is most common in the lumbo-sacral region; after this in the cervical and thoracic. It is often associated with other malformations, such as umbilical hernia, etc., and the infants seldom survive.

2. *Myelocystocele.* This form consists in arrested development of the vertebral arches and dura mater. It appears in the third week of embryonic life, at a time when the medullary groove has closed to form the neural tube, and the epiblast has grown over it. Hydrops of the central canal causes bulging of the posterior part of the spinal cord through the gap in the vertebral arches, giving rise to a tumor-like swelling of the spinal cord covered by the soft parts. The substance of the spinal cord soon undergoes degeneration and can only be identified by the presence of ciliated cylindrical epithelium on the inner surface of the cavity (the remains of the ciliated epithelium

of the central canal of the spinal cord). In the external coverings of myelocystocele there is often lipoma, myxoma, lymphangioma or teratoma. The tumor has a wide base and is covered with normal skin, which is thickened at the base of the tumor. Sometimes small depressions are present in the skin caused by the remains of amniotic bands (Fig. 144). The tumor is of soft consistence, and fluctuation is always present. The fluid contents of the tumor can be completely reduced by pressure, as there is direct communication with the central canal, and also with the subarachnoid space. By pressing on the tumor the transmission of fluid pressure can be felt at the fontanelle.

Myelocystocele is often combined with hydrocephalus. Paralyses are rare, as the motor nerves are not displaced by the malformation; at the most there may be pes varus or valgus on one side, due to the tumor being situated unsymmetrically more to one side of the middle line, and thus dragging on a motor nerve. However, extensive myelocystocele of the lumbo-sacral region may cause paralysis of the bladder and rectum. Trophoneurotic disorders are common. Sometimes paralysis occurs at a later age, the tumor increasing gradually in size and dragging on the spinal cord and nerves. Defective bone formation is often associated with myelocystocele—absence of vertebral bodies, unilateral defects in the vertebral laminæ, absence of ribs or patella, scoliosis, etc.

3. *Meningocele.* According to recent observations meningocele can only occur in places where the spinal cord is absent (*von Bergmann*). In this condition there is defective formation of the vertebræ and dura mater, so that the pia mater protrudes posteriorly, inclosing the filum terminale. In this way a pedunculated swelling is formed, covered by normal skin, which may attain the size of a child's head as the amount of cerebro-spinal fluid in the sac increases.

Paralysis only occurs when the meningocele is large, and is then generally of limited extent. There is sometimes abundant hair on the summit of the swelling. Fluctuation is always present, but there is only slight diminution on pressure. The space in the bone is generally smaller than in myelocele. Meningocele occurs most often in the sacral region.

Spina Bifida Occulta, according to the most recent observations, is a form of meningocele which becomes ruptured and undergoes spontaneous healing under the skin. The pressure of the cicatrix may cause disturbances which are not noticed till the child grows older.

Differential Diagnosis. Myelocele, when the vasculo-medullary zone is very extensive, may sometimes be mistaken for cavernoma. In rare cases where epidermization of the second zone leads to cicatrization of the third zone, myelocele may be mistaken for a myelocystocele in which the skin has become cicatrized after ulceration. In such cases diminution of the tumor on pressure points to myelocystocele. In lipoma, lymphangioma and teratoma there is no diminution in the tumor on pressure unless there is a myelocystocele underneath it; which, however, is often the case. Meningocele may be mistaken for myelocystocele when it is not situated in the sacral region (where the spinal cord is absent). It may also be mistaken for sacral tumors, dermoids and teratomata.

The prognosis is not unfavorable in myelocystocele and meningocele provided other malformations are absent and the infant has a strong constitution.

Treatment. In myelocele a radical operation is useless, because by removal of the cystic sac the spinal cord is divided and unites with the cicatrix. Reduction of the infected vasculo-medullary zone by operation always leads to meningitis. Palliative

treatment, by puncture of the sac, is all that can be done in these cases.

Myelocystocele, owing to its covering of intact skin, is more suitable for operation. In this case the operation is similar to that for hernia. The sac, consisting of degenerated spinal cord, is exposed by an incision through the skin, dissected down to the bone, ligatured and removed. The cleft in the bone is repaired by a plastic operation. The sac is often covered by a fatty tumor which also requires removal. Removal of the sac after ligature is not dangerous in these cases, as it consists only of functionless degenerated spinal cord. Meningitis sometimes follows these operations, but most cases recover and may grow up.

Meningocele offers the best chances for operation. The sac is opened and the nerves replaced in the vertebral canal. The sac is then ligatured and removed and the space in the bone closed by suture of the soft parts, or by bone grafting. The prognosis is good after these operations.

In spina bifida occulta with disturbances due to pressure of the cicatrix, the latter may be removed and the space in the bone repaired.

The development of hydrocephalus, which may occur after operation on all forms, is an unfavorable sign.

Fig. 143 shows a myelocele of the lumbo-sacral region. The tumor is situated symmetrically in the middle line and has a wide base. At the base the skin is thickened (first zone); the second zone (epithelio-serous) shows numerous ramifying vessels; the third zone (vasculo-medullary) is not typical and resembles the second zone, owing to epidermization of the latter (cf. Fig. 142). It only differs from the second zone in its bluish color. The diagnosis of myelocele depended on the absence of diminution on pressure, and the presence of paralysis of the bladder

and rectum, and pronounced pes varus of both feet. Death occurred soon after birth.

Fig. 144 shows a myelocystocele situated in the lumbar region, and covered with normal skin. A small depression in the surface is due to amniotic adhesions. Under the skin is a mass of fatty tissue, while a deep cystic tumor could be felt more deeply situated. The latter could be almost completely emptied by pressure. There were no motor or sensory disorders present, and no other malformations. The X-rays showed a small cleft in one of the vertebral arches situated a little to one side of the middle line. The superficial fatty tumor was removed and found to be a myxolipoma. The myelocystocele was then separated down to the bone, ligatured and removed. The gap in the vertebra was closed by transplantation of a piece of bone from the iliac crest. Microscopic examination showed the pressure of cylindrical epithelium in the inner wall of the cyst, thus confirming the diagnosis.

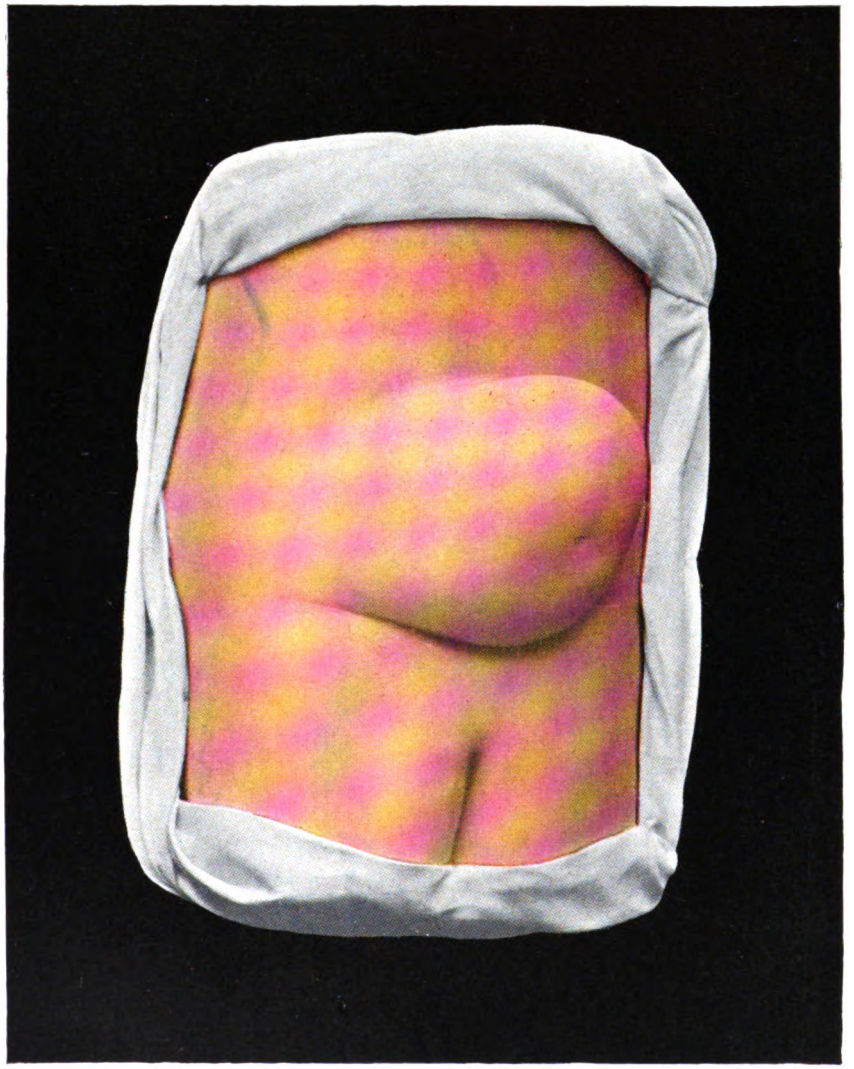

Fig. 144. Myelocystocele – Myxolipoma.

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PES VARUS

Pes varus may be congenital or acquired. The congenital form may be caused by arrested development, or may be secondary to pressure caused by amniotic adhesions, etc. Congenital pes varus is common in connection with myelocele, and is due to paralysis of the nerves, as already explained. Acquired pes varus occurs in rickets, and as the result of poliomyelitis which causes paralysis of the pronators and dorsal flexors of the foot. The chief effect takes place at the midtarsal joint and consists in supination, plantar flexion, internal rotation and adduction. Changes also occur in the astragalus and os calcis, especially in long-standing cases. These changes can be seen by the X-rays. There is also shortening of the muscles, tendons, fascia and ligaments, especially shortening of the tendo Achillis (talipes equino-varus). Decubital ulcers may form on the outer border of the foot.

Treatment. In congenital clubfoot treatment should be begun as early as possible, by repeated manual correction to the normal position, followed by fixation in an over-corrected position by means of plaster of Paris bandages. In sucklings, thin strips of cotton bandages soaked in mastic solution (turpentine 15, mastic 12, resin 28, alcohol (90 per cent.) 180, ether 20) may be used, applied to the foot and leg so that the foot is fixed in the over-corrected position. This treatment should be kept up for six months, after which elastic traction may be applied to the foot for another six months. In older children manipulation must be performed under an anæs-

thetic. After the ninth month preliminary tenotomy of the tendo Achillis is necessary, before the foot can be brought into the proper position. To prevent relapse boots should be worn with the sole raised on the outer side, but care must be taken to avoid producing flat foot. In pes varus due to poliomyelitis tendon transplantation may be performed. Old-standing cases of clubfoot in adults require osteotomy, or sometimes more extensive operations such as disarticulation.

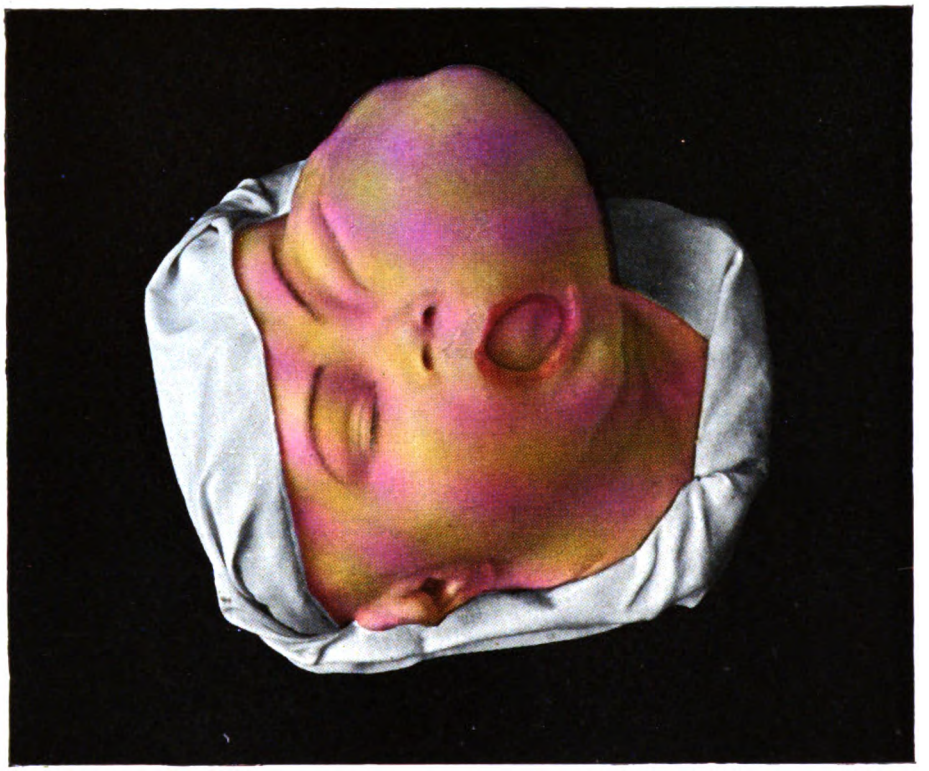

Fig. 146. Teratoma monogerminale.

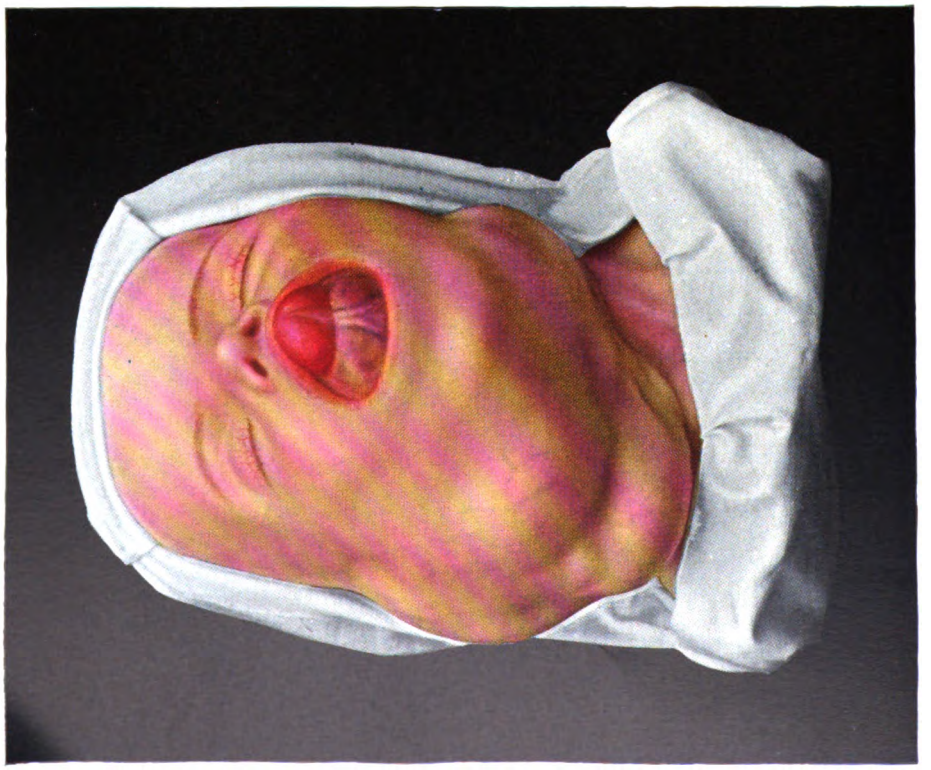

Fig. 145. Lymphangioma congenitum multiplex.

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Lymphangioma

LYMPHANGIOMA CONGENITUM CYSTICUM MULTIPLEX

(Congenital Multiple cystic lymphangioma)

Plate CXVII, Fig. 145.

The term lymphangioma should be limited to those tumors in which there is a new formation of lymphatic vessels. A number of growths have been included in the term lymphangioma which are only formed of dilated lymphatics, without any new lymphatic vessels. Microscopic examination is therefore important in these cases. Clinically, we distinguish simple, cavernous and cystic lymphangiomas; also single and multiple. In the great majority of cases the growths are congenital. All three forms are often present in the same patient. Lymphangiomas may occur in the skin, but more often in the subcutaneous tissue; also between the muscles and in the subserous tissue. The term simple lymphangioma is wrongly applied to lymphangiectases, which form lobulated growths covered by thickened skin and occur on the head, trunk and extremities. Simple lymphangioma occurs most commonly in the tongue or lips as a circumscribed growth. The skin or mucous membrane is always somewhat thickened and adherent to the growth. The isolated circumscribed form of simple lymphangioma may be mistaken for other kinds of tumor, but transitional stages to cavernous lymphangioma are often found, the diagnosis of which is easier.

Cavernous lymphangioma is always a diffuse for-

mation, of soft consistence and always united with the skin or mucous membrane over it. The tumor can be gradually diminished by pressure, as the cavernous spaces, filled with lymph and lined with epithelium, communicate with the neighboring lymphatic vessels. Cavernous lymphangioma forms a painless, diffuse, slow-growing tumor with a smooth surface and irregular borders. When they are visible under the skin or mucous membrane they have a pale-green color, in distinction to the reddish-blue color of cavernous hemangioma. They occur most often in the cheeks, tongue and lips, giving rise to enlargement of these parts, known as macromelia, macroglossia and macrocheilia. They are generally congenital, or appear soon after birth. Cavernous lymphangiomas also occur in the neck, causing a dimpled swelling of the skin by their numerous processes, which extend in all directions (Fig. 145). As already mentioned, lymphangiomas may be situated over encephaloceles or myelocystoceles. Gradual atrophy of the bones may be caused by the pressure of extensively progressing lymphangiomas.

Cystic lymphangioma occurs in the subcutaneous or intermuscular tissue, most often in the side of the neck (Fig. 145). It is composed of large, cystic cavities lined by epithelium and containing whitish or brownish fluid (cystic hygroma). Cystic lymphangioma is almost always congenital and is characterized by its slow growth, which may cease after some years. The skin is unchanged and can be raised from the tumor. Fluctuation is present, but there is no diminution of the tumor on pressure. Extensive lymphangioma of the neck may be dangerous from pressure on the trachea. Besides the neck, the growths may also occur in the axilla, the popliteal space, the bend of the elbow, the groin and the sacral region. Infants with congenital lymphangioma sometimes show other malformations, and are often incapable of life.

Differential Diagnosis. Simple lymphangioma which occurs in the form of a small, soft, circumscribed tumor, may be mistaken for fibroma, lipoma or hemangioma. Cavernous lymphangioma can only be mistaken for hemangioma, as no other tumor diminishes on pressure. It differs from hemangioma in its greenish color and in the nature of its contents. Cystic lymphangioma, when it occurs in the form of an isolated unilocular cyst, may be mistaken for various tumors; in the neck, for blood cyst, branchial cyst, lipoma or dermoid.

The prognosis of lymphangioma is, on the whole, not unfavorable, on account of its limited growth and occasional spontaneous resolution.

Treatment. Circumscribed lymphangioma can be excised. In diffuse cavernous lymphangiomas (macrocheilia, macroglossia, macromelia) cuneiform excision may be performed. The introduction of magnesium may be tried, to cause thrombosis and shrinking of the tumor. After this extirpation is easier and infection through a lymph fistula, which so often occurs after the usual operation, is avoided. Cystic hygroma is best treated in this way. Radical operations should not be performed unless the child is in good condition. Puncture and injection of tincture of iodine are unsafe measures, while lymph fistula often remains after incision and plugging. Lymph fistulas must always be removed by a radical operation, on account of the danger of infection through them. Lymph fistulas, which occur from injury to the thoracic duct after extensive extirpation of the breast, can be healed by plugging with acetate of aluminium.

Fig. 145 shows a congenital tumor involving the lower part of the right cheek, the whole of the right side of the neck and the greater part of the left side of the neck. The skin was unchanged and

movable over the tumor. On examination, it was found to be a multilocular cystic tumor. There was no diminution on pressure. The tumor also extended to the floor of the mouth, so that the tongue, which also contained a lymphangioma (macroglossia), was displaced upwards. The greenish surface of the cyst was visible under the mucous membrane of the mouth, so that the diagnosis of congenital multiple cystic lymphangioma was made. On account of the situation of the tumor on both sides of the neck in the submaxillary, submental and parotid regions, the case might be mistaken for an affection first described by *Mikulicz*, in which there is symmetrical enlargement of all the salivary glands and glands of similar structure in the head and neck. In this case, however, there was no change in the lachrymal glands, which are usually affected in *Mikulicz's* disease; also there was a characteristic lymphangioma in the tongue, which is absent in *Mikulicz's* disease. The swelling of the floor of the mouth on each side of the froenum of the tongue resembles a ranula. The latter is a cystic formation arising most commonly in the duct of the sublingual gland, more rarely from the incisive gland situated on the inner surface of the lower jaw in the middle line.

Teratomata

TERATOMA MONOGERMINALE (*Monogerminal teratoma*)
Plate CXVII, Fig. 146.

Teratomas may be bigerminal or monogerminal. In bigerminal teratoma there is a true double formation —a fetus within a fetus. In monogerminal teratoma there is perverted development in one embryo, and all the tissues are derived from one embryo only. The latter includes all kinds of mixed tumors which are formed of all three embryonic layers (epiblast, mesoblast and hypoblast). Dermoid cysts, which are formed by all three embryonic layers, belong to the teratomata. A distinction between monogerminal and bigerminal teratomata is not always possible, and is of little clinical importance. Teratomas are rare on the whole, and are always congenital. They are most often found in the buccal cavity, where they may be mistaken for naso-pharyngeal polypi (Fig. 25). They also occur in the face, neck and coccygeal region, and have been observed in the mediastinum and abdominal cavity. They may attain enormous dimensions, and have then an irregular, uneven surface. The consistence also varies, some parts being cystic, others soft and others hard. Teratomas often form encapsuled tumors. They may cause extensive destruction by pressure on the neighboring parts. A distinction between teratomata and teratoid mixed tumors is clinically impossible. Diagnosis in many cases is only made after examination of the extirpated tumor.

Differential Diagnosis. Teratomas which appear as large, congenital tumors can generally be recognized by the above-mentioned characteristics, especially by their situation in the embryonic fissures. Diagnosis is assisted by the X-rays which may reveal bones and teeth, which are often present in teratomas. Teratomas occurring in the thorax, abdomen and pelvis, especially when they do not assume a tumor growth till later years, can often only be diagnosed by operation.

Treatment. Teratomas have been successfully removed both in children and in adults. Extensive teratomas (Fig. 146) cannot be removed by operation. The presence of other deformities, such as spina bifida, and the feeble condition of the infants often renders operative treatment impossible.

Fig. 146 shows a teratoma of the left side of the face, almost as large as the fist, involving the left orbit and almost the whole of the buccal cavity, and covered by livid, movable skin. It was covered by a connective-tissue capsule. Further examination showed that it arose from the base of the skull, but did not communicate with the cranial cavity. The tumor was soft and fluctuating in some places, hard in others. Examination by the X-rays showed the presence of a piece of bone, which was afterwards found to be part of the upper jaw. Further examination showed that the tumor consisted of neuroglia, neuroepithelium and cysts lined with epithelium. As it consisted of epiblastic products only it must be regarded as a monogerminal tumor which, in this case, originated from a separated portion of the epiblast. This view is supported by the fact that the tumor developed in a region (base of the skull) where separation of the epiblast is possible. On the other hand, it appears far-fetched to consider the tumor as a bigerminal teratoma (fetus within

fetus by inclusion) simply because of its large size at birth.

There were no other malformations present except mutilation of the right ear. Death occurred soon after birth.

DUCTUS OMPHALO-MESENTERICUS PERSISTENS

(*Persistent Omphalo-mesenteric Duct*)

Plate CXVIII, Fig. 147.

The omphalo-mesenteric duct, or vitelline duct, is the communication between the alimentary canal and the umbilical vesicle or yolk-sac. It usually disappears about the eighth week of fetal life. In some cases this duct may persist and is then known as *Meckel's* diverticulum, which arises from the small intestine about ten inches above the ileocæcal valve. This diverticulum may lie free in the abdominal cavity, where it may cause intestinal obstruction by becoming entangled with the intestines; or it may become attached to the umbilicus, or extend a short distance into the umbilical cord. In the latter case it may become opened after birth when the umbilical cord has separated, thus giving rise to an umbilical fistula, discharging fæces from the umbilicus when the whole length of the duct is open as far as the intestine. When the intestinal end of the duct is closed, the remainder may persist as a small fistula discharging mucoid secretion; or it may become dilated into cystic formations.

In umbilical fistula there is a red globular swelling with a small depression at its apex, situated at the navel. The surface of the swelling is formed by mucous membrane. A probe can be passed through the depression as far as the small intestine, and the greater part of the fæces are discharged through the fistula, causing inflammation of the skin surrounding the navel. Death often occurs from prolapse of the small intestine.

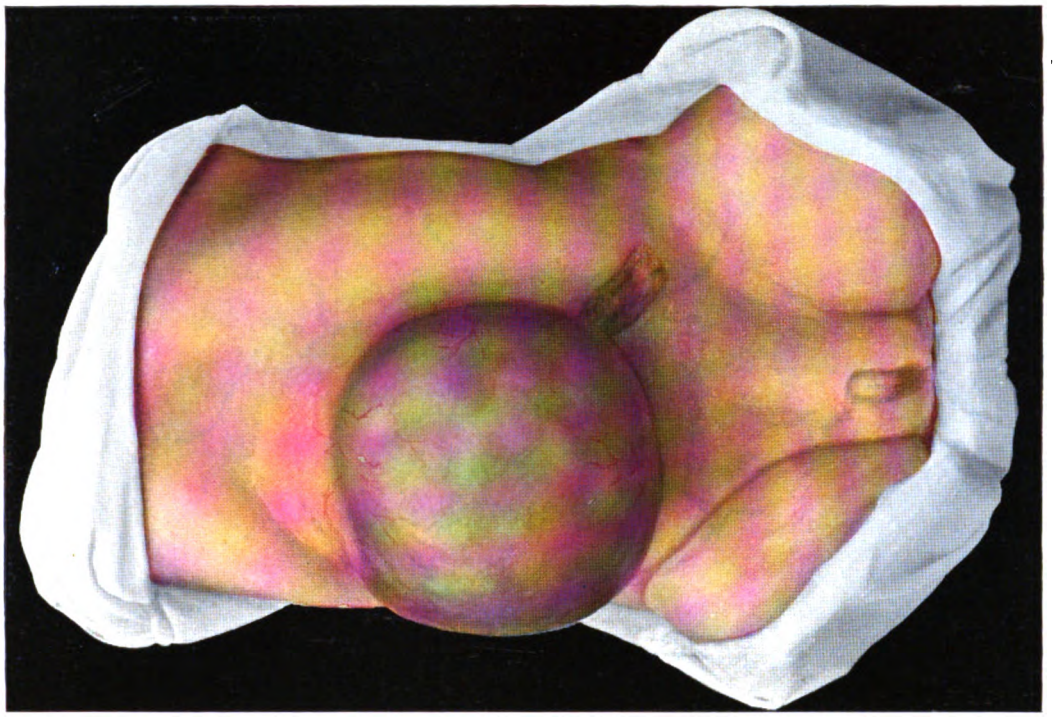

Fig. 148. Hernia funiculi umbilicalis congenita.

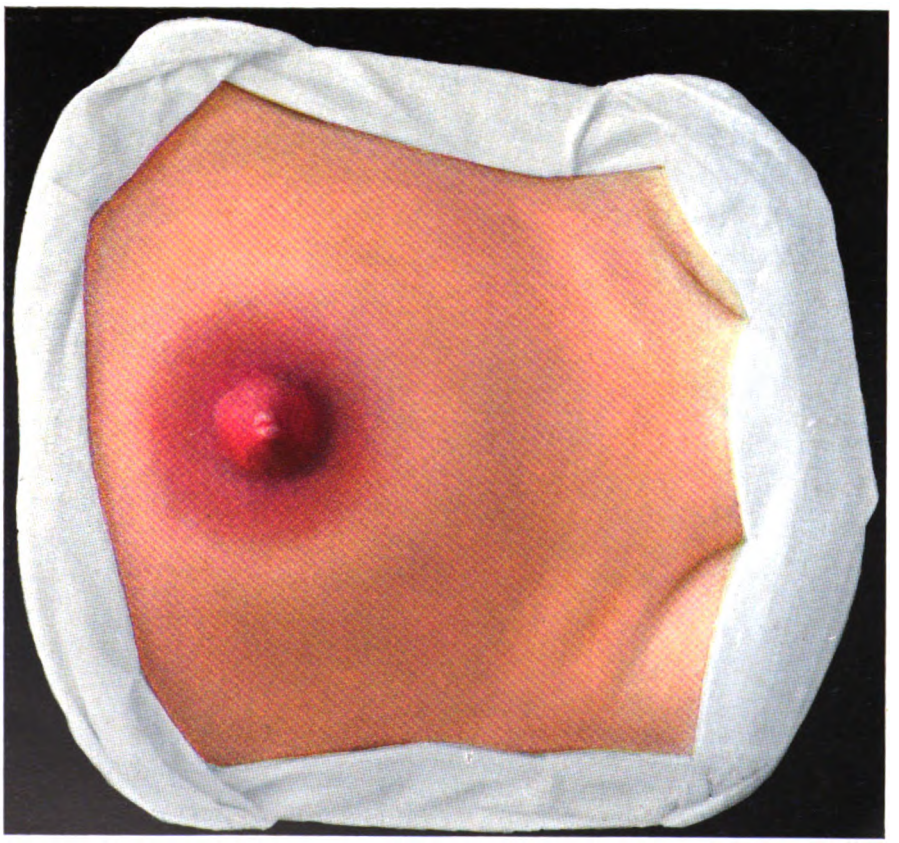

Fig. 147. Ductus omphalo-mesentericus persistens.

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Differential Diagnosis. Infection of the navel with the formation of granulation tissue may resemble the above condition. Other fistulas may also occur in the umbilicus. The urachus, which represents the remains of the communication between the bladder and the allantois in fetal life, may remain open and form a fistula at the umbilicus. Normally the urachus becomes obliterated and forms the median ligament of the bladder. Fistula of the urachus is diagnosed by discharging urine. Like fistula of the vitelline duct, fistula of the urachus usually appears after separation of the umbilical cord. The nature of the fistula is not always determined by probing; a more certain method of diagnosing fistula of the vitelline duct is by feeding with powdered charcoal, which then appears at the navel. The diagnosis can sometimes be made by chemical and microscopical examination of the secretion. Tuberculosis of the intestine, actinomycosis, peritonitis, empyema of the gall bladder, injuries of the bladder, and dermoids may all give rise to fistula at the navel.

Treatment. Fistula of the vitelline duct can sometimes be prevented by discovering the condition before tying the umbilical cord. The cord is then thicker than usual at its base. The end of the duct can then be reduced and the cord tied further away from the navel.

In cases of complete fistula leading to the intestine laparotomy is necessary, with resection of the diverticulum and suture of the intestine. Fistula of the urachus must be separated down to the bladder and removed, and the bladder sutured.

Fig. 147 shows a case of complete fistula of the vitelline duct. The infant was in a bad condition from prolapse of the gut, evacuation of fæces from the navel, and inflammation of the surrounding skin. Laparotomy was performed but the operation was unsuccessful.

HERNIA FUNICULI UMBILICALIS CONGENITA

(*Congenital Umbilical Hernia*)

Plate CXVIII, Fig. 148.

Congenital umbilical hernia must be regarded as a malformation, and forms a large tumor, containing intestine and often also the liver. It is often associated with various forms of spina bifida, or with ectopia of the bladder. Cases of extensive umbilical hernia are due to arrested development causing incomplete closure of the abdominal walls. Umbilical hernia may also be acquired. In this case the abdominal walls are closed, the umbilical ring is small, the hernia is smaller and more cylindrical, and the contents consist of small intestine. Acquired hernia may be so small as to be overlooked at birth, and may then be included in the ligature of the umbilical cord. The base of the cord should, therefore, always be examined to see if it contains intestine.

Congenital umbilical hernia forms a large globular swelling in the region of the navel (Fig. 148). The surface is destitute of cutaneous covering and shows the greenish-yellow remains of the amnion. The remains of the umbilical cord is generally seen at one side of the swelling. In rare cases epidermization takes place at the borders; more commonly the swelling ruptures from pressure, with consequent prolapse of the viscera and death from peritonitis.

Differential Diagnosis. Both the congenital and the acquired forms of umbilical hernia are so characteristic that they cannot be mistaken for any other condition.

Treatment. The occurrence of symptoms of intestinal obstruction, or threatening perforation of the sac indicate immediate laparotomy, with excision of the sac, reduction of its contents and closure of the abdominal walls. In some cases the viscera are adherent to the sac and require separation. Reduction of the visceral contents is sometimes difficult or even impossible, especially when the liver is contained in the sac. If operation is not urgent it may be postponed till the child is stronger, the sac being supported by bandaging in the meantime.

Acquired umbilical hernia may occur during the first month after birth, as the umbilical ring takes several weeks to close completely. Anything which causes the infant to cry may be an exciting cause for hernia, also straining from phimosis, etc. Many cases become cured without treatment. Non-operative treatment consists in placing a metal disk wrapped in plaster over the umbilical ring, after reduction of the hernia, and bringing the skin of the abdomen together over it by means of plaster. The disk must be larger than the hernial opening. Small openings may be closed in this way after nine months' treatment. Larger openings with separation of the recti muscles above the umbilical ring require laparotomy. In older children, especially girls, this should always be performed. The operation consists in extirpation of the whole umbilical ring and suture of the abdominal walls with wire.

In Fig. 148 the hernial sac contained the intestine and liver, which were reduced with great difficulty, so that the abdominal walls when sutured were under great tension. The infant died soon after the operation.

AMPUTATIONES AMNIOTICÆ (*Amniotic Amputations*)
Plate CXIX, Fig. 149.

Malformations of the extremities include *amelus* and *phocomelus*. In amelus the extremities are absent or only represented by stumps. This condition may affect all four extremities, both arms or legs, or one arm or leg. In phocomelus there is arrested development of the proximal segments of the arms or legs, or of all four extremities. The hands or feet are then situated directly on the trunk. Some of these cases attain adult age, and one has been known to live to sixty-two. [Several such cases were among *Barnum's* freaks.]

The so-called spontaneous amputations of various parts of the extremities are caused by pressure of amniotic bands or the umbilical cord. The ends of the amputations are then pointed. In other cases there is not complete amputation but constriction, resulting in deep, circular grooves extending to the bone (Fig. 149). In spite of the depth of the grooves, the circulation remains normal, but there is often elephantiasic thickening from lymphatic congestion. In some cases the bones are constricted, as shown by the X-rays. The remains of the amniotic bands are often present in the constricted places.

Other malformations, also due to tightness of the embryonic membranes, are synechia of the fingers (webbed fingers), hare-lip, cleft-palate, transverse fissure of the cheek, and fissure of the tongue.

Treatment. When the constricted parts are functionless they should be amputated. Elephantiasis may be treated by cuneiform excision.

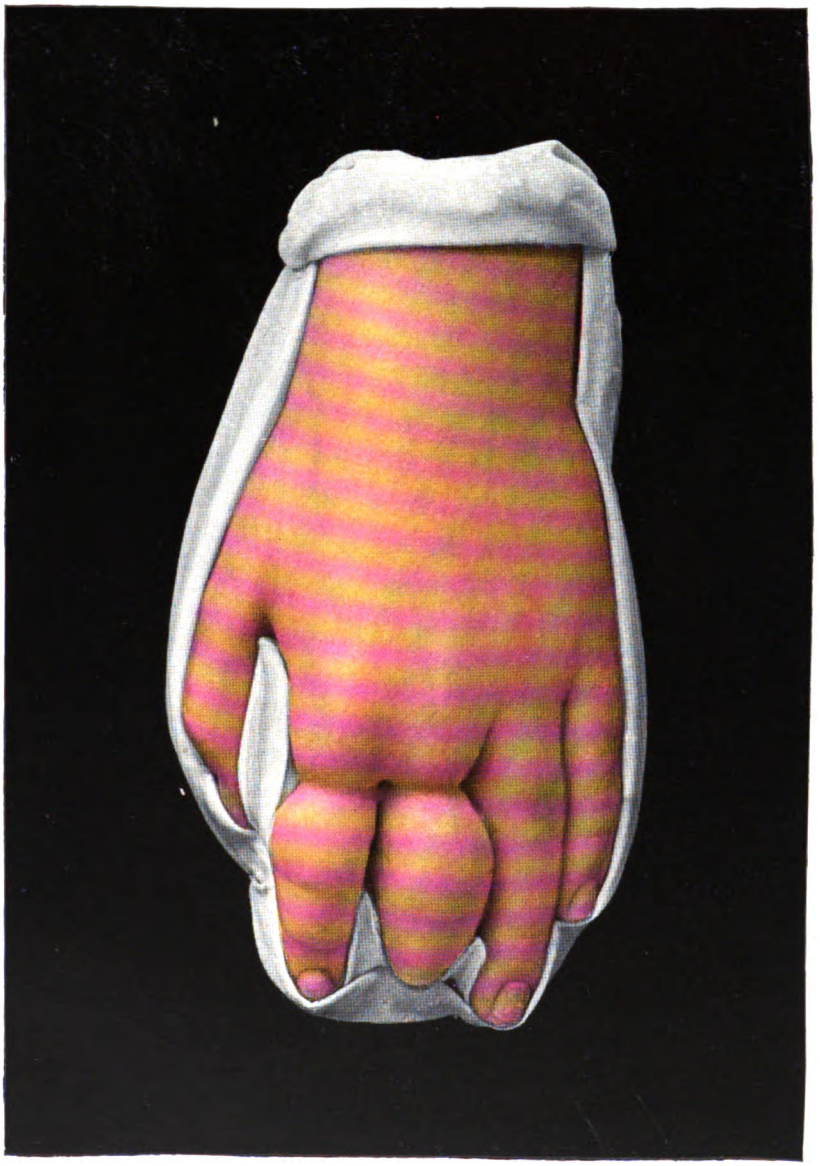

Fig. 149. Amputationes amnioticae.

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In Fig. 149 the function of the fingers was normal so that no operation was necessary. In this case there was also hare-lip and cleft-palate, which were operated upon.

AKROMEGALIA (*Acromegaly*)
MAKROMELIA (*Macromelia*)
MAKROGLOSSIA (*Macroglossia*)
Plate CXX, Fig. 150.

The term Acromegaly is applied to a condition in which there is enlargement of the terminal portions of the body—the hands, feet, nose, cheeks, tongue and ears. The enlargement affects all the tissues (true giantism) and does not appear till after the termination of the period of growth, thus differing from congenital giantism. In some cases there is increased growth of hair, and curvature of the vertebral column. The disease causes considerable disfigurement of the face. It generally appears between the twentieth and fortieth years and may remain stationary. In many cases there is, first of all, hypertrophy of the bones of the hands, feet and face.

The disease has been attributed to changes in the pituitary body (hypertrophy, adenoma, sarcoma, cyst); to changes in the thyroid gland, pancreas, genital glands; to persistence of the thymus; to nervous influence, since nervous disorders have been observed in the hypertrophied extremities; also to a congenital condition. The most probable of these is enlargement of the pituitary body, which can be demonstrated by widening of the sella turcica, shown by the X-rays. Large tumors of the pituitary body may press on the optic nerve and nerves of the ocular muscles.

The prognosis is not unfavorable, as severe disturbances only occur after the disease has existed for many years.

Differential Diagnosis. Partial giantism, which also begins in the hands and feet, differs from acro-

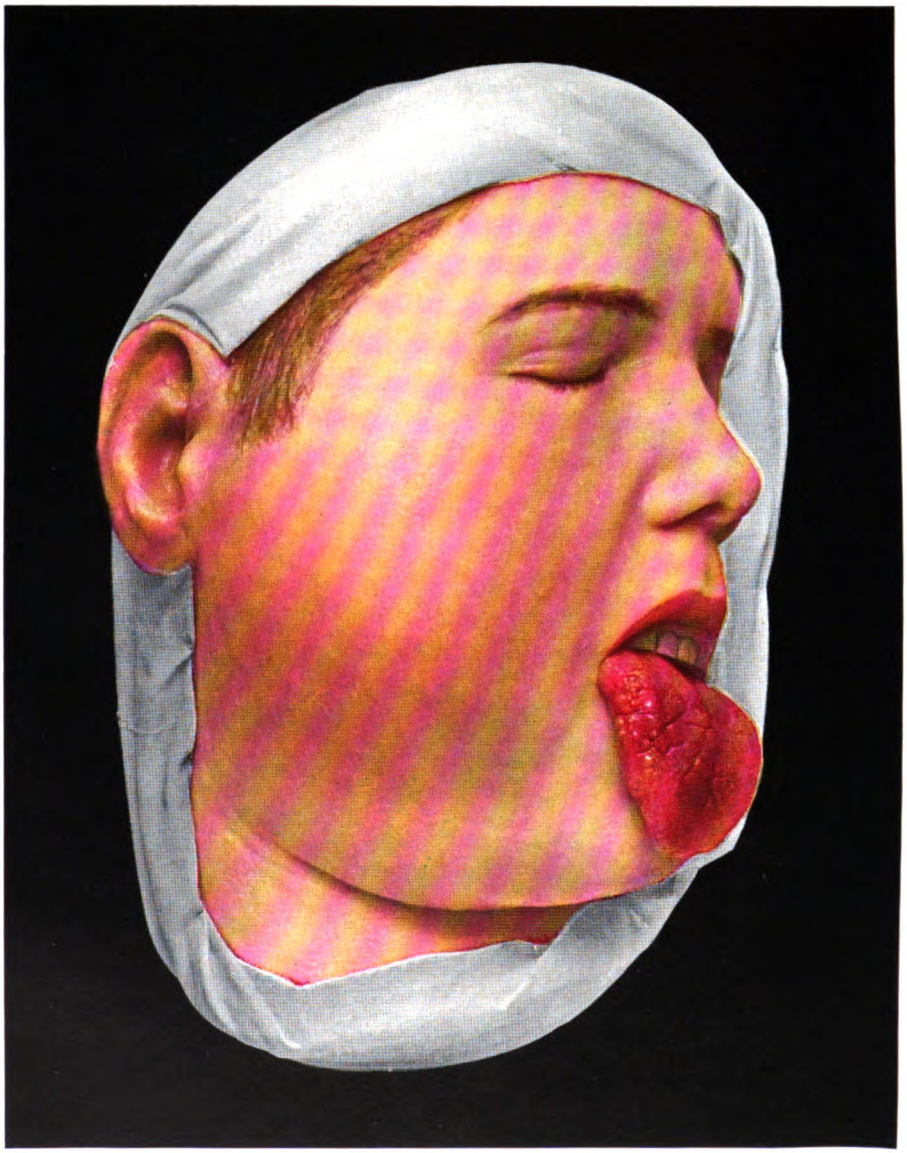

Fig. 150. Akromegalia — Makromelia — Makroglossia.

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megaly by being congenital. In leontiasis ossea there is enlargement of the bones, while the soft parts are more often atrophied. Acromegaly affecting one extremity only might be mistaken for osteitis deformans, or for chronic osteomyelitis, as there may be lengthening of the bone in both these diseases. Acromegaly differs from elephantiasis in the presence of enlargement of the bones, which can be shown by the X-rays. Acromegaly commencing in the face might possibly be mistaken for tumor of the upper maxilla, but there is usually early hypertrophy of the cheeks (macromelia), lips and tongue (macroglossia), and of the hands and feet.

Treatment. Thyroid extract and extract of pituitary gland have been recommended. Tumor of the pituitary gland may be removed by operation. Extensive enlargement of the soft parts may be diminished by cuneiform excision.

Fig. 150 shows marked hypertrophy of the right side of the face. The right ear is considerably larger than the left, and there is hypertrophy of all the tissues of the cheek. The right side of the tongue is enlarged, somewhat resembling cavernous lymphangioma, but differing in being unilateral. X-ray examination showed unilateral enlargement of the upper and lower maxillary bones. The fingers and toes on the right side had increased in size for some years. The X-rays showed widening of the sella turcica indicating the presence of a tumor of the pituitary body. As the patient suffered no trouble from the disease, he refused operation.

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